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POSTPARTAL CARDIOMYOPATHY

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Postpartal heart disease has been described in an increasing number of publications since the first report by Virchow[1] in 1870. The literature has been reviewed in 2 recent papers by Rosen[2] and by Benchimol *et al.*[3] The most important papers on this subject have been those by Hull and co-workers,[4,5] Gouley *et al.*,[6] Musser *et al.*,[7] Meadows,[8] and Benchimol *et al.*[3]

Analysis of this literature shows that many of the cases described as postpartal had their beginning during or even before pregnancy; many had an aetiology referable to specific disease, especially pre-eclamptic toxaemia of pregnancy, nephritis, and hypertension of other origin. Some had a non-specific inflammatory myocarditis, and some were so complicated by pulmonary thrombosis or embolism as to leave doubt whether the postpartal state was significant in the aetiology or not. Benchimol *et al.*[3] considered that, on careful assessment, cases described as occurring postpartum could be allocated to aetiological types, so that doubt concerning the identity of a specific postpartal heart disease remains. The majority of authors emphasize the haemodynamic changes that occur in pregnancy and continue after delivery, which may lead to an uncovering of heart disease in the puerperium.

The present study is of 13 female patients, considered to be suffering from the cardiomyopathy of Africans seen in the southern part of Africa, whose illness began postpartum. No claim is made that they represent a specific postpartal heart disease; the implication of the occurrence is that whatever factors cause this cardiomyopathy may be exaggerated in the postpartal state. The patients were carefully selected to exclude the possibility of other aetiology; all but 2 had their onset sufficiently far removed from the delivery to render the haemodynamic changes of pregnancy and the puerperium irrelevant (i.e. 1 month or more). The other 2 with a shorter interval were so similar that their inclusion seemed justifiable.

AFRICAN CARDIOMYOPATHY

African cardiomyopathy is now generally considered to be of 2 broad groups: the endomyocardial fibrosis of North, East and West Africa, and the cardiomyopathy of Southern Africa which is largely free from this fibrosis.[9,10] Clinical studies of the Southern African type were first reported by Gillanders,[11] and pathological studies were reported by Higginson *et al.*[12] and Becker *et al.*[13] who emphasized the endocardial changes that do occur, though they are very different in degree from the gross changes of the northern group. Whether these 2 groups are fundamentally different, or differ only in some superadded factor, remains to be seen.

The cardiomyopathy seen at King Edward VIII Hospital is of the southern type. No cases corresponding to the endomyocardial fibrosis of North, East and West Africa have been seen. The clinical presentation is a familiar one, and the cases to be described were such orthodox examples that the possibility of the postpartal cases being of different aetiology from the usual cardiomyopathy is considered minimal.

MATERIAL

Seventeen female patients have been seen in heart failure in whom no primary non-myocardial cause for the heart failure could be found. Strict criteria for inclusion were adhered to in these cases of cardiomyopathy. Any patient whose diastolic blood pressure was known to be more than 90 mm.Hg at rest either at previous incidental admission, at delivery, after response of the heart failure therapy, or at subsequent follow-up, was excluded. Hypertensive retinopathy of Grade 1 or more excluded the patient. Any patient with signs or symptoms suggesting pulmonary embolism at or near the beginning of the illness was excluded, as was any with primary pulmonary dysfunction. Any patient with more than slight albuminuria, or with granular casts or cells in the urine, was excluded. Evidence of a purely left ventricular lesion on radiography disallowed the patient. The only cardiac murmurs allowed were ordinary soft ejection murmurs, and mitral or tricuspid pansystolic murmurs of Grade 2/4 or less, which disappeared with recovery from heart failure and decrease in heart size. Any significant fever, leucocytosis or anaemia caused exclusion. Finally, occasional cases of tuberculous pericarditis occur in Africans in whom a thick layer of granulation tissue leads to a radiological cardiomegaly, with low amplitude, congestive cardiac failure and the clinical features of cardiomyopathy. To exclude these, any doubtful case was submitted to right atrial catheterization and angiography to demonstrate the thickness of the atrial wall. In this way 2 cases were excluded.

The strictness of the criteria in all probability disallowed several true cases from admission to the series, but they allow a corresponding security of diagnosis.

Of the 17 cases, 13 had their first symptoms in the puerperium. In 3 there was no relationship to pregnancy or the puerperium; in 1 the onset was at the 6th month of pregnancy. It is the 13 cases arising postpartum that form the substance of this report.

CASE DETAILS

History of the Illness

Some of the relevant data are given in Table I. The age range extends over nearly the whole child-bearing period; 6 of the patients were 30 years or more, 7 less than 30 years of age. The length of history in all but 4 cases was a month or less; in 1 patient it was most acute and caused admission after only a day's illness. In all these patients there was

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TABLE I. SOME RELEVANT DATA CONCERNING THE PATIENTS IN THIS SERIES

	Patient	Age in years	Duration of symptoms	Interval between delivery and onset	No. of previous deliveries	Birth	B.P. on admission (mm. Hg)
1	G.A.	20	1 week	6 weeks	2	Single	130/95
2	P.N.	34	3 months	3 months	5	Single	120/90
3	D.N.	39	1 month	5 weeks	3	Single	120/75
4	P.S.	31	3 weeks	1 week	5	Single	140/110
5	A.D.	34	3 weeks	5 months	5	Single	130/85
6	B.N.	23	1 day	5 weeks	?	Twins	150/100
7	A.H.	27	4 weeks	1 month	4	Single	130/90
8	M.Q.	26	3 weeks	1 week	5	Single	150/110
9	S.M.	30	4 months	1 month	11	Twins	100/?
10	F.H.	30	1 week	3 months	?	Single	130/90
11	F.M.	28	3 days	1 month	?	Single	120/90
12	R.D.	29	3 months	6 weeks	4	Single	125/105
13	Sh.N.	23	14 months	4 months	1	Single	110/60

progressively severe dyspnoea on exertion, and in 9 there was a cough which produced either nothing or small amounts of sputum, occasionally blood-tinged. Nine had noticed swelling of the legs, and 5 complained of a tight, oppresive feeling in the chest when dyspnoeic, which in 3 could be severe enough to be called a pain.

The interval between delivery and onset of symptoms varied from 1 week to 5 months, and bore no relation to the duration of symptoms before admission. In this interval all patients felt entirely well and were fully mobile. All were breast-feeding their infants on admission, and in all this was stopped soon after. The number of previous deliveries was known in 10 cases and showed a wide scatter.

The dietary history, as is usual in this African population, was poor and largely carbohydrate. A simple method of quantitating the value of the diet in this population is by the number of days per week on which meat is eaten. On this basis, the diet when meat is taken less than once per week is Grade 0. If meat is eaten once, the diet is Grade 1; if twice, Grade 2; if 3 times, Grade 3, and if more than 3 times, Grade 4. Of the present patients, 1 took a Grade 0 diet, 4 were Grade 1, 4 were Grade 2, and 4 were Grade 3. In a control healthy group of 100 postpartal women, 4% took a Grade 0 diet, 12% Grade 1, 21% Grade 2, 17% Grade 3, and 46% Grade 4. There is no good evidence from these figures that the patients took any poorer a diet than the general population from which they came.

In 11 cases (Table I) the preceding delivery was of a single infant; in 2, twin infants were born.

Physical Examination

The findings on physical examination were fairly uniform. All except 1 patient appeared well nourished, the exception having dry lack-lustre skin and hair. They were all in congestive heart failure on admission; the heart was typically large with a hypodynamic pulsation, felt laterally and parasternally, the pulse was small and fast, and the hands cold. There was in all a diastolic gallop sound, heard best in the mitral area, which was timed on phonocardiography in 6 patients. The time after the aortic second sound was 0·08 seconds in 1, 0·14 in 3, 0·16 and 0·18 seconds in 1 each. An early gallop sound has been recorded as well in male cases proved at autopsy, and cannot be used as a differential diagnostic point for a pericardial lesion. A pansystolic murmur due to functional atrio-ventricular valve incompetence was heard in 3 patients. The murmur disappeared with rest and decrease in heart size; it was mitral in 2, and tricuspid in 1.

Treatment and Progress

The progress of the patients and their treatment was variable. Four were kept at bed rest without any medication, on a meat-free, carbohydrate diet similar to their own for an initial period of 4 days. They were then given, by injection, 100 mg. each of pyridoxine, pantothenic acid, aneurine and nicotinamide daily for 3 days; and by mouth 60 mg. of riboflavine daily for the same period. Thereafter they were followed for a further 3 days on no therapy, as in the initial period of observation. Finally digitalis with or without a diuretic was administered. During all this time accurate daily assessments were made of pulse rate, blood pressure, jugular venous pressure, heart size, urine output, weight, and the degree of oedema and hepatomegaly. In none of these 4 was there any response to the vitamins. Two other patients begun on this regime had to be treated, from the third and fourth days, because of dangerous deterioration of the clinical state.

In 2, digitalization by 18 gr. of digitalis leaf in 3 days followed by a maintenance dose of 2 gr. daily did not cause any significant change in the subjects within 5 days of the commencement of this therapy. The giving of a mercurial diuretic and restriction of salt led to rapid and marked improvement in 11 of the 13 patients. The other 2 remained refractory; 1 was eventually discharged free from heart failure, only to return after 4 weeks, again in severe decompensation. The other improved so slowly over 3 months that she was finally discharged, still in failure, on her own insistence. She was still in severe heart failure 5 months after discharge. It is noteworthy that the 2 refractory patients were those who had produced twins.

Of the 11 who improved well, 2 were apparently cured at the time of discharge, all signs and symptoms having disappeared; 1 of these was still well 1 month after discharge, having taken digitalis. Unfortunately she then failed to attend again and could not be traced, so that it is not known how far she remained in health without therapy. The other did not re-attend and could not be traced.

Six were followed for periods up to 1 year; 5 needed continued digitalis, 1 being completely symptom-free on this regime. The patient who did not need therapy was also quite free from symptoms after 3 months without treatment, but she had clear radiological evidence of persisting cardiomegaly. No patient in this series was found to be free from signs of heart disease when followed up.

The remaining 3 patients, who were followed up for less than a month, continued to show signs and need therapy, and subsequently failed to re-attend and were untraceable.

TABLE II. SERUM PROTEIN AND TURBIDITY FIGURES IN 11 PATIENTS AND IN CONTROLS

	Serum albumin (g. per 100 ml.)	Serum globulin (g. per 100 ml.)	Serum alkaline phosphatase (King-Armstrong units)	Zinc turbidity (units)	Thymol turbidity (units)
Present series	3·0 (2·4 - 3·7)	3·9 (2·9 - 5·4)	8·1 (4 - 11)	16·4 (8 - 31)	5·4 (2 - 11)
Control series	2·7 (1·5 - 3·7)	3·3 (2·2 - 4·2)			

Investigations

According to the criteria for admission to the series, none of the patients had significant anaemia, leucocytosis or urinary abnormality.

Serum-protein and turbidity tests were performed in 11 patients. The mean and range of values is given in Table II, together with those available of a control healthy postpartal female population of 27 cases.[14]

Since the serum-albumin level probably reflects the protein nutritional state there does not seem from these figures to be any greater deficiency than in the normal population.

Liver biopsies were performed in 4 patients. In 1 of the biopsies there were a few binucleate cells; in another a slight amount of fat was present. The other 2 showed nothing worth comment, and in general all 4 showed no evidence of the damage that is associated with chronic malnutrition in these African patients.

The toxoplasma complement-fixation test was performed in 3 patients and was negative in all.

Pressures were measured in the pulmonary circulation in 2 patients after clinical improvement. The right ventricular pressures were 30 and 40 mm.Hg systolic, and 0 and 5 mm.Hg diastolic respectively; the mean right atrial pressures were 5 mm.Hg in both, and the mean left atrial pressures (wedged pulmonary artery) were 12 and 20 mm.Hg respectively.

Chest X-rays taken on the first admission, when the patients were in congestive heart failure, showed large hearts and marked pulmonary congestion. On screening, both ventricles appeared to be involved in the dilatation, and their movement in contraction was small. As the patients improved the heart size came back towards normal. In the least successful it remained large, in the most successful the lung fields cleared and the heart size, though persistently abnormal, was considerably reduced.

Twelve-lead electrocardiographic examination was made in all patients on from 2 to 6 occasions, except in 1 patient where only a single tracing was obtained. Analysis of the leads on admission shows that the axis was deviated to the left in 6; the electrical position was horizontal in 6, vertical in 2, and mid-position in the remainder. The transition from dominant S wave to dominant R wave in the chest leads occurred at V_2 - V_4 in 9, V_4 - V_5 in 4. The maximum amplitude of P (which was in lead II in 12 cases and in lead I in 1 case) was 1 mm. mean, range 0·25 - 2 mm. The mean duration of the P wave was 0·08 seconds, range 0·06 - 0·1 seconds. It was biphasic in V_1 in 5 cases, bifid in V_2 in 2 cases and in V_4 in another. The mean P-R interval was 0·135 seconds, range 0·12 - 0·16 seconds. Small q waves, never greater than ½ mm. in depth, were present in V_5 and V_6 in 3, accompanying a similar wave in lead II in 2. There were similar q waves in leads I and aVL in 1 case. The mean corrected Q-T interval was 0·439 seconds, with a range, 0·379 - 0·514 seconds. The sum $S(V_1)+R(V_5)$ was 31·4 mm. mean, range 13·5 - 52 mm.; in only 3 was the sum greater than 35 mm. The sum $R(V_1)+S(V_5)$ was 1·8 mm. mean, range 0 - 7·5 mm. The maximum potential in the augmented limb leads had a mean of 6·6 mm., range 2·5 - 11 mm. Five patients had a maximum of less than 5 mm. The QRS duration in 11 patients in lead II was 0·064 seconds mean, range 0·05 - 0·08 seconds. The other 2 patients had a left bundle branch block pattern with a QRS duration of 0·12 and 0·14 seconds. This pattern disappeared in 1 patient in the 6th month after her discharge; she was then free from symptoms on digitalis. T waves were inverted over the left ventricular surface leads in 6, over the right in 1, over both left and right surfaces in 4. The maximum height or depth of the T wave in standard and unipolar limb leads was 1·1 mm. mean, range 0·25 - 2 mm. In the chest leads the maxima had a mean of 4·3 mm., range 1 - 10 mm. In subsequent tracings the following changes took place: in 3 the T wave inverted or deepened in leads V_1 - V_3; in 1 in leads V_4 - V_6. In 1 the T wave, initially inverted in leads V_4 - V_6, became shallower there. Two patients showed a progressive anti-clockwise rotation, and 1 rotated clockwise.

DISCUSSION

The clinical history and findings and the results of the investigations are all typical of Southern African cardiomyopathy. The cases have not differed from male cases except in the age incidence and the prognosis. The mean age of these 13 postpartal cases was 29, and of the non-postpartal female cases, 31 years. In a comparable series of 31 male cases, personally studied, the mean age on admission was 41, and only 4 cases were below the age of 30. A possible implication is that the exaggeration of the aetiological factors in the puerperium is responsible for the earlier onset in females. So far no cases have been seen in nulliparous African females. It is of particular interest that the 2 patients who had produced twins were the only 2 completely refractory to therapy. The tendency for twin pregnancy to be associated with postpartal heart failure has been recorded before and is reviewed by Meadows[8] and Rosen.[2]

The difference in prognosis is to some extent a matter of impression, because of small numbers and poor follow-up. Two postpartal patients out of 13 were discharged completely free from symptoms and signs, clinical or investigatory. Only 1 African male out of 31 studied, and many more seen casually, responded to this extent. Two further postpartal patients, 1 of whom was still on treatment, were symptom-free at follow-up, but continued to show cardiomegaly. This degree of response occurred in only 2 out of the 31 male patients. Two postpartal patients were completely resistant to treatment: refractoriness in males is sufficiently common to have caused 6 out of 31 to die, and a further 3 to be discharged after several months of hospital regime, still in heart failure.

The better prognosis of the postpartal group was seen also in the 4 female patients whose illness started apart from the peurperium. One of these was apparently cured at the time of discharge but has not been seen without therapy; the other 3 were much improved but had persistent signs. These numbers are too small to resolve the point whether the puerperal factors are sufficiently reversible to make the prognosis better than in those females in whom these factors were not operative. However, in general, the puerperal factor alters the age incidence and the prognosis compared with male cases. A possible interpretation of the postpartal occurrence of an illness common in this population is that a postpartal process occurs which merely precipitates heart failure, the cardiomyopathy may be fundamental and the postpartal process transitory. Since the nature of both of these is unknown, one can go no further with reasonable speculation.

It is difficult to know how far the puerperal cardiomyopathies described in people of European origin are related to the present cases. On the criteria for inclusion in this series no individually reported European case could be included. On the other hand several investigators have reported cases in American negroes which may be included,[4,9,15] and Keely[9] has briefly mentioned the occurrence in this country. No cases have been seen in the Asian patients of this hospital, although their numbers are the same as Africans in the general population served by the hospital. Such racial differences frequently imply a

dietary difference. The failure of the 4 patients tested to respond to B vitamins shows only that there was no acute deficiency of these substances. Similar results were seen in 18 male cases submitted to the same trial and have been reported for this type of cardiomyopathy by Gillanders.[11] The diet of the present cases, though deficient in protein over probably their whole life-time, was no worse than that of a control sample. Moreover, except in 1 case no signs of other malnutrition were present; such signs, especially those of pellagra, are not uncommon in the hospital population, yet no tendency for these pellagra patients to have cardiac symptoms has been noted. Neither clinical beri-beri nor response to aneurine has been observed in any patients.

A simple protein-deficiency factor is rendered unlikely by the serum-protein findings and by the histological findings in the liver in 4 patients. Normal liver histology has been reported in a non-postpartal case by Higginson *et al.*,[12] who also reported absence of fibrosis in 2 others. In the 31 cases of cardiomyopathy in males that were studied, the appearance of the liver was known either by biopsy or autopsy in 15. In 6 of these there was no fibrosis, and in 4 no abnormality at all. Fibrosis may, therefore, be absent in the male cases. It has not been seen in the postpartal cases. Since it is thought to be an invariable accompaniment of prolonged protein deficiency in these adult patients, the findings argue against this deficiency factor in postpartal cardiomyopathy. Hull and Hidden[5] and Gillanders[11] have reported findings for the serum proteins similar to the present figures.

Meadows[8] also considered that there was no nutritional factor in his cases. Sodeman,[15] however, stated that nutritional deficiency was common in the patients who displayed the picture of postpartal heart failure, and 2 of the cases of Benchimol *et al.*,[3] grouped as non-specific myocarditis, had evidence of vitamin deficiency.

Although there is evidence that toxoplasmosis is a common infection in this country,[16] the complement-fixation test did not reveal this aetiology for the cardiomyopathy in the postpartal cases, and has given similar results in male cases. The myocarditis reported by Paulley *et al.*[17] has not therefore been observed.

The pressure measurements from the pulmonary circulation in 2 cases merely confirm the clinical evidence of biventricular failure, in contrast to the pulmonary hypertensive cases described by Abrahams.[18] Similar findings have been made in 6 non-postpartal cases.

The radiological features in these cases are the same as those previously described in this condition[11] and as recorded in European cases of cardiomyopathy.[19]

Electrocardiography shows, as the dominant variation, T-wave flattening and inversion, often with progressive changes. There is a tendency to small voltage QRS deflections, and a prolonged Q-T interval. Abnormal Q waves are notably absent. The findings in similar cases have been reported by Hull and Hafkesbring,[4] Gillanders,[11] Becker *et al.*[13] and Meadows.[8]

Hull and Hafkesbring[4] reported 1 postmortem examination in a 16-year-old negress. The findings, briefly reported, indicate a different picture, a myocarditis rather than a non-inflammatory cardiomyopathy. Moreover the illness began before delivery. It may be that this was in fact a different type of case, so that the pathological picture remains uncertain. The reported appearances in European cases have been quite unlike the African cardiomyopathy, the commonest description being of severe focal scarring. Higginson *et al.*[12] have reported the findings in the African variety, which consist of interstitial oedema, a fine interstitial fibrosis, hypertrophy of muscle fibres with hyperchromatic nuclei and a subendocardial fibrosis that, if present, is patchy, rarely diffuse, and usually not wider than the space occupied by about 6 muscle fibres. In occasional cases the band of fibrosis may be 10 or more fibres in diameter and macroscopically obvious. Since no deaths from postpartal cardiomyopathy have been seen here, final evidence as to the identity with non-postpartal cases is lacking, but there seems no good reason to doubt this identity.

SUMMARY

1. Thirteen patients are described in whom heart failure presumed due to 'African cardiomyopathy' developed postpartum. Electrocardiographic and other investigatory results are presented.

2. Nutritional factors as studied by dietary history, clinical appearance, vitamin replacement, plasma-protein levels, and liver biopsy gave no evidence for any specific nutritional deficiency.

3. Twin deliveries occurred in the 2 cases which were most refractory to therapy.

4. The postpartal cases showed greater improvement than a comparable male series, but none reversed completely.

5. It is postulated that some unknown postpartal process precipitates heart failure in patients with African cardiomyopathy, itself of unknown aetiology, and that the postpartal process may well be transitory.

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VAN DIE REDAKSIE : EDITORIAL

VERDERE FAKTORE BY SPONTANE MISKRAAM

Bevrugting en die instandhouding van swangerskap berus op die vereniging van 'n gesonde ovum en 'n gesonde sperma, die bestaan van 'n goeie buis- en baarmoederomgewing, en 'n geskikte biochemiese omlysting. As enigeen van hierdie faktore versteur is, kan spontane miskraam of voortydige geboorte ontstaan. In 'n onlangse redaksionele artikel[1] het ons verwys na sommige van hierdie faktore, veral wat betref sekere genetiese aspekte, en ons het ook syfers in hierdie verband aangehaal.

Daar is voorheen gemeen dat defekte van die kiemplasma van die belangrikste oorsake van spontane miskraam is. Dit word nou egter al meer duidelik dat daar verskillende toksiese, metaboliese, en endokrienfaktore is wat ook 'n uitwerking op die gamete kan hê; ook dat siektetoestande, metaboliese en voedingsversteurings en ernstige endokrienafwykinge 'n uitwerking het op die biochemiese omgewing wat die ingeplante ovum in stand hou. Meganiese faktore soos binnebaarmoederlike fibroïede, aangebore abnormaliteite van die uterus en 'n verswakte baarmoedermond kan natuurlik ook 'n uitwerking hê op die ovum. Endokrienstudies, wat berug is omdat hulle so ingewikkeld en duur is om uit te voer (en wat in elk geval net deur ervare tegnici aangedurf behoort te word), het aangetoon dat daar endokrienabnormaliteite voorkom by pasiënte met miskrame. Hierdie afwykinge mag sonder behandeling regkom — 'n feit wat aandui dat onverklaarde, maar tydelike, faktore ook 'n rol kan speel. As ons nou die feite waarmee ons bekend is by ons vermoedens voeg, dan wil dit voorkom of endokrienondersteuning van swangerskap hoofsaaklik bepaal word deur chorioniese gonadotrofien, progesteroon, estrogeen, en die hormone van die skildklier.

Choriomiese gonadotrofien, wat deur die ovulêre trofoblast afgeskei word, bereik 'n hoë vlak in die gestel gedurende die eerste trimester, en dit hou skynbaar die corpus luteum in stand wat progesteroon voortbring totdat die plasenta dié funksie kan oorneem. Tydelike afnames in die vlak van gonadotrofien lei nie altyd tot miskraam nie (wat ook die geval is met voortdurende lae vlakke). Hierdie feite gee aanleiding tot die vermoede van defekte van die ovum nog voor inplanting plaasgevind het, of ongenoegsame prikkeling van die endometrium voor en na inplanting.

Progesteroon berei die endometrium, (waarop estrogeen alreeds 'n invloed gehad het) voor vir inplantasie, dit hou die buitenste vrugvlies in stand, hou die baarmoederspier in 'n rustende toestand sodat die swangerskap bestendig kan word, en verseker 'n genoegsame bloedtoevoer vir die swangerskap. Die corpus luteum is die belangrikste bron van progesteroon gedurende die eerste trimester, waarna dit vervang word deur die plasenta. Hierdie feit mag 'n verklaring wees waarom verwydering van die corpus luteum in vroeë swangerskap nie altyd die swangerskap versteur nie. Hierdie hormone kan op sy beste bepaal word deur sy metaboliese produk in die urien - pregnandiol, alhoewel die bepaling slegs by benadering gedoen kan word. Daar moet egter weer aangetoon word dat voortdurende lae vlakke van die hormone beskou moet word as voorlopers van miskraam.

Estrogeen word afgeskei deur die volwasse follikel, dan die corpus luteum en uiteindelik die ovulêre trofoblast teen ongeveer die 90ste dag van swangerskap. Dit berei die endometrium voor vir die progesteroon-respons, help dan om die buitenste vrugvlies in swangerskap in stand te hou deur die bloedvat-voorsiening van die uterus te verbeter en groei van die uterus te bevorder sodat dit die groeiende embrio kan bevat. Estrogeen-vlakke word bepaal deur gebruik te maak van metaboliete. Lae produksie is ongewoon; 'n balans tussen estrogeen en progesteroon is waarskynlik noodsaaklik vir normale swangerskap. Die spesifieke verhouding tussen die twee hormone is nog nie vasgestel nie.

Die rol van die skildklier by bevrugting en instandhouding van swangerskap is ook nog nie baie duidelik nie. Dit is noodsaaklik vir die normale kiemselle sowel as vir die instandhouding van die groeiende embrio. Jodium wat gebonde is aan die serum-proteïen, is 'n sensitiewe indeks van tirioïed-funksie; dit word min of meer verdubbel kort na bevrugting plaasgevind het, en bly op daardie vlak dwarsdeur swangerskap. As die vlak daarvan nie styg nie, volg miskraam gewoonlik.

Hierdie feite verteenwoordig 'n betreklik vereenvoudigde beeld van die endokrien-agtergrond van bevrugting en swangerskap. Dit is duidelik dat die toestand van sake beïnvloed mag word deur ander bestaande faktore, aangesien die harmoniese verloop van swangerskap deur 'n veelvuldigheid van faktore bepaal word, almal waarvan nog nie bekend is of ten volle verstaan word nie.

1. Van die Redaksie (1961): S. Afr. T. Geneesk., **35**, 125.

CONFUSION IN NOMENCLATURE

The pharmaceutical industry has made many important contributions in recent years in providing drugs which will decrease or terminate illness and promote longer and happier lives. Twenty-five years ago only a few drugs that were really effective were available to the medical profession. Today there are scores of potent agents widely used in general practice and in all the specialist branches of medicine. The discovery and development of these drugs is undertaken in the many research laboratories of the pharmaceutical industry.

If a new compound is a therapeutic success, similar methods of manufacture are usually evolved by other

firms, and very soon the new drug is advertised under many different proprietary names. Alternatively, a series of compounds may be introduced with very similar therapeutic actions, but differing only slightly in their chemical constitution. These all become advertised under different names. The result is confusion in the nomenclature of new drugs. After an interval a new drug may be accepted for the pharmacopoeia, and is given an official name. For obvious reasons it becomes difficult to establish the popular use of the official name.

Practitioners are well aware of the confusing nomenclature, and pharmacists are also perplexed by the multiplicity of similar-sounding names. There are many examples that could be quoted but a few will suffice. Among the tranquillizers there are triflupromazine and trifluoperazine, perphenazine and prochlorperazine. There are digoxin and digitoxin; raudixin and redoxon; sigmagen and sigmamycin; romical and romilar; librium and libratar; and metreton and meticorten. There must be even better examples of drugs with different actions and indications whose names are very similar. It is surprising that accidents do not occur frequently from the use of the wrong drug. It is to the credit of doctors, nurses, and pharmacists that mistakes occur very infrequently.

It would be a blessing to both prescribers and dispensers of drugs if the manufacturers would relegate their brand names to second place and market their products as penicillin (Smith) or penicillin (Jones). Unfortunately this method of nomenclature is unacceptable to commercial firms, although it is applied to insulin preparations.

As the output of new preparations increases, the search for new names becomes a big problem. One firm in the USA has 'commissioned a machine to produce a dictionary of forty-two thousand nonsense words of an appropriate scientific look and sound'. An official said 'Thinking up names has been driving us cuckoo . . . A good trade name carries a lot of weight with doctors . . . there are enough names in the new dictionary to keep us going for years . . . We don't yet know what proportion of names is unpronounceable . . . how many are obscene . . . how many objectionable on other grounds of good taste. "Godamycin" would be a mild example[1].'

It is therefore clear that a difficult situation confronts the practising physician. Perhaps the doctors have themselves to blame for this. In many countries attempts have been made and progress achieved in obtaining uniformity on a national level. The World Health Organization has attempted to coordinate these efforts at an international level, and has published lists of proposed international non-proprietary names for drugs.

It is obvious that there is much to be done, and a difficult task lies ahead to avoid the confusion which exists in the multiplicity of names for the same preparations and the similarity of names for different preparations.

1. New Yorker, 14 July 1956.

TYLOSIS PALMARIS ET PLANTARIS FAMILIARIS ASSOCIATED WITH CLINODACTYLY

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Tylosis palmaris et plantaris familiaris (hereafter referred to as tylosis) is a familial ectodermal anomaly of the palms and soles producing marked hyperkeratosis. It is to Thost[25] and Unna[27] that credit must be given for the original description of the disease. Since then numerous reports have appeared in the literature. The most comprehensive review of the whole subject, however, is that of Cockayne.[3] Several workers have noted the rare association between tylosis and other conditions. Among them are Howel-Evans *et al.*[9] who discussed the association of this disease with carcinoma of the oesophagus. Modern genetic studies have focussed attention on the co-existence of familial conditions. In view of this it was felt necessary to study, in more detail, the family of a patient who presented with a myocardial infarction and a family history of tylosis.

A family of 59 members through 5 generations was investigated. Wherever possible they were personally interviewed. Where this was not practicable a first-hand collaborative history was taken from several other family members. Particular attention was paid to the presence of tylosis and other ectodermal anomalies, arterial degenerative disorders, dysphagia, and other abnormalities. In those deceased the cause of death was ascertained.

CASE REPORT

Mr. C.P., aged 42 years, lorry driver, was admitted to the Johannesburg General Hospital on 12 September 1959 with a myocardial infarction following a period of angina of effort. Typical hyperkeratotic lesions of the soles and palms, with extension of the lesion to the dorsal surfaces of the index and little fingers, were present. Similar small areas of horny thickening of the skin were noted over the extensor aspects of both elbows, overlying the tendo Achillis, and to a lesser extent in the dorsal ankle crease. An interesting feature was anhydrosis of the affected areas. The patient gave a good description of tylosis in other members of his family. The condition apparently became evident when he began to crawl at the age of 6 months but had not really troubled him until he became engaged in manual labour. He noted that his skin was much improved in a humid atmosphere and that use

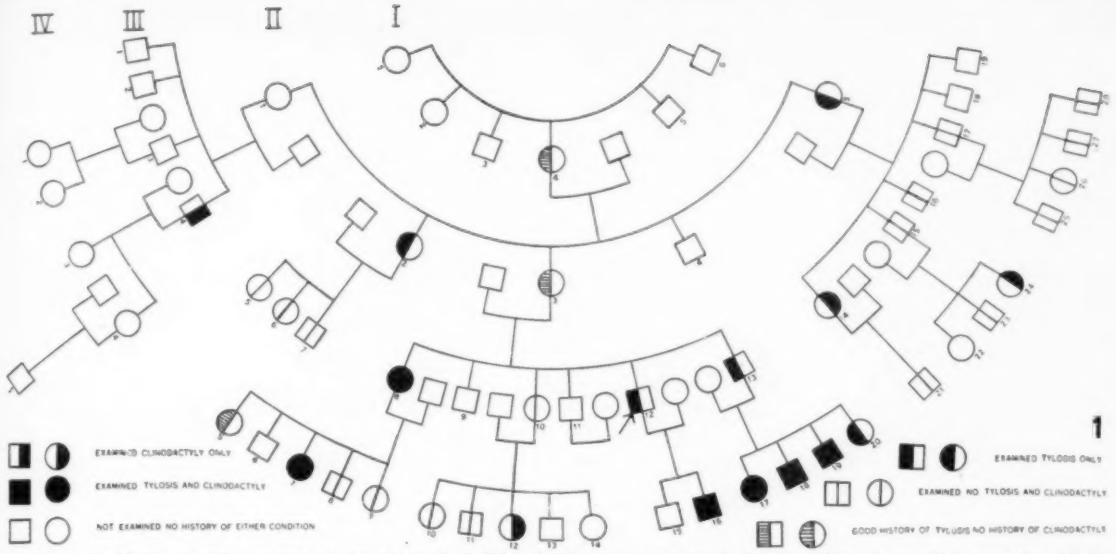

Fig. 1. Family tree of patient, showing 4 generations (I, II, III and IV) and one member in fifth generation. Arrow points to patient (III 12).

TABLE I. SUBJECTS WHO WERE PERSONALLY EXAMINED OR HAD HISTORY OF TYLOSIS

Family member†	*Sex*	*Age (years)*	*Examined or not*	*Living or deceased*	*Presence of tylosis*	*Distribution and severity of lesions*							*Presence of clinodactyly*	*Sweating of hands and feet*
						Soles	*Palms*	*Dorsum elbow*	*Tendo Achillis*	*Knee*	*Dorsum foot*	*Medial malleoli*		
I 4	F	95	N E	D	Yes	—	—	—	—	—	—	—	—	—
II 2	F	50	E	L	Yes	++	++	—	—	+	—	—	—	Increased
II 3	F	51	N E	D	Yes	—	—	—	—	—	—	—	—	—
II 5	F	64	E	L	Yes	+++	+++	+	—	—	—	+	—	Decreased
III 4	M	50	E	L	No	—	—	—	—	—	—	—	Yes	Normal
III 5	F	11	E	L	No	—	—	—	—	—	—	—	No	Normal
III 6	F	9	E	L	No	—	—	—	—	—	—	—	No	Normal
III 7	M	7	E	L	No	—	—	—	—	—	—	—	No	Normal
III 8	F	38	E	L	Yes	++	+++	—	—	+	+	—	Yes	Decreased
III 10	F	33	E	L	No	—	—	—	—	—	—	—	No	Normal
III 12	M	42	E	L	Yes	+++	+++	+	+	—	+	—	No	Decreased
III 13	M	35	E	L	Yes	++	+++	—	+	—	—	—	No	Decreased
III 14	F	24	E	L	No	—	—	—	—	—	—	—	Yes	Normal
III 15	M	41	E	L	No	—	—	—	—	—	—	—	No	Normal
III 16	M	18	E	L	No	—	—	—	—	—	—	—	No	Normal
III 17	M	42	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 5	F	17	N E	L	Yes	—	—	—	—	—	—	—	—	—
IV 7	F	14	E	L	Yes	++	++	—	+	—	—	+	Yes	Decreased
IV 8	M	13	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 9	F	7	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 10	F	14	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 11	M	12	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 12	F	10	E	L	No	—	—	—	—	—	—	—	Yes	Normal
IV 16	M	5	E	L	Yes	+++	+++	+	+	—	+	—	Yes	Decreased
IV 17	F	12	E	L	Yes	++	++	—	+	—	—	+	Yes	Normal
IV 18	M	10	E	L	Yes	++	++	+	+	+	+	+	Yes	Decreased
IV 19	M	7	E	L	Yes	++	++	—	+	—	—	+	Yes	Normal
IV 20	F	3	E	L	Yes	++	++	—	—	—	—	—	No	Normal
IV 21	M	1	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 23	M	9	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 24	F	5	E	L	No	—	—	—	—	—	—	—	Yes	Normal
IV 25	M	10	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 26	F	6	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 27	M	8	E	L	No	—	—	—	—	—	—	—	No	Normal
IV 28	M	2	E	L	No	—	—	—	—	—	—	—	No	Normal

F = female, M = male, NE = not examined, E = examined, D = deceased, L = living, + = slight, ++ = more pronounced, and +++ = severe.
† For numbers refer to Fig. 1.

of the hands, especially in cold dry weather, resulted in gross thickening, cracking and even bleeding of the palmar skin. Except for the presence of tylosis and evidence of acute myocardial infarction, general examination of the patient was essentially normal.

Family

In the 59 members of the family there were at least 14 with tylosis (9 female, 5 male). Of the 32 members personally examined, 11 were affected; a good description was obtained regarding 3 others (Fig. 1 and Table I). In all affected cases the age of onset was in the first year of life. In 10 of the subjects clinodactyly was noted. In this condition the little finger is curved inwards towards the other fingers. No other abnormality of skin or skeleton was noted nor were there any abnormalities of hair or teeth. Besides the patient, 2 (II5, III11) of the 33 living members had sustained myocardial infarctions. No other members gave a history of cardiovascular disease. There was no history of dysphagia or buccal leukoplakia and as far as could be ascertained no one had died of oesophageal carcinoma. Of the 7 in whom the cause of death was known (I4, II1, II3, II4, III9, III18, and III19), 3 were due to cardiac disease (I4, II1, and II3). None of the 7 had died of malignant disease.

DISCUSSION

Tylosis

Much confusion exists in the literature regarding the terminology of the group of hyperkeratoses. Tylosis palmaris et plantaris has the following synonyms:[23] keratosis palmaris et plantaris, hyperkeratosis palmaris et plantaris, ichthyosis palmaris et plantaris, keratoderma palmare et plantare, and symmetrical keratoderma.

The condition has been described as rare.[22] In Northern Ireland the incidence was calculated to be 1 in 40,000.[24]

Histopathologically,[19] the skin usually shows considerable hypertrophy of all its layers, more especially the stratum corneum, which is grossly thickened. The stratum granulosum is normal in appearance and there is no change in the stratum spinosum. Occasionally there is flattening of the papillary body. These papillae may be increased fivefold in depth. The dermis is unaffected, except outside the area of horny thickening or where fissures are present, when mild inflammatory changes may be noted. The sweat glands and their ducts may be hypertrophied. The histopathological section in the present case is shown (Fig. 2) compared with a normal section (Fig. 3).

Clinically tylosis is rarely manifest at birth and is usually not recognized until the third or fourth month. In exceptional cases the onset may be delayed until the age of 6 years.[19,23] The lesions are bilateral, symmetrical, and situated almost exclusively on the palms and soles (Figs. 4 and 5) either of which may be predominantly involved. Occasionally the hyperkeratosis is noted on the dorsum of the hands, feet and phalanges. In some cases it is present on the extensor surfaces of the elbows, over the knees and about the ankles.[15,19] The nails may be involved and become thickened and opaque and are raised up from the nail bed by a horny accumulation beneath them. This complication may result in more severe symptoms than those produced by the skin lesions.

The distribution is determined to a large extent by physical factors such as pressure and friction. Thus, in infants, the lesions will be on the knees; on beginning to walk the feet are most involved in the weight-bearing areas of the soles; the manual labourer exhibits gross

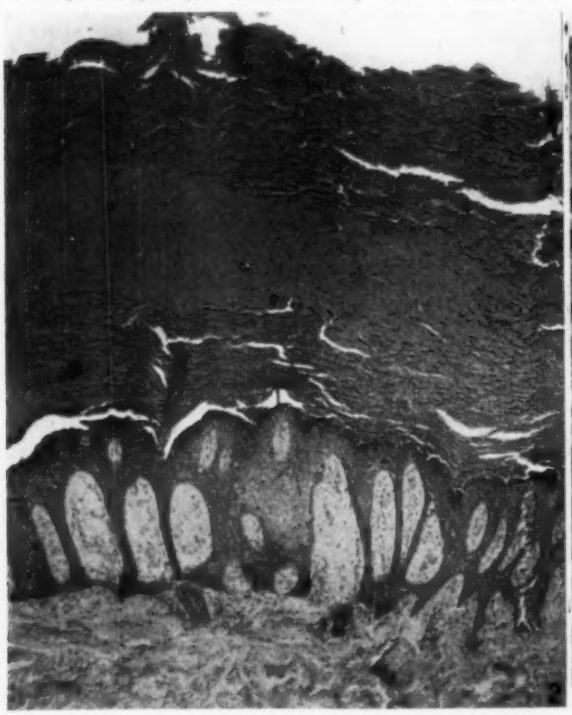

Fig. 2. Low-power section of hyperkeratotic palmar skin (× 45).

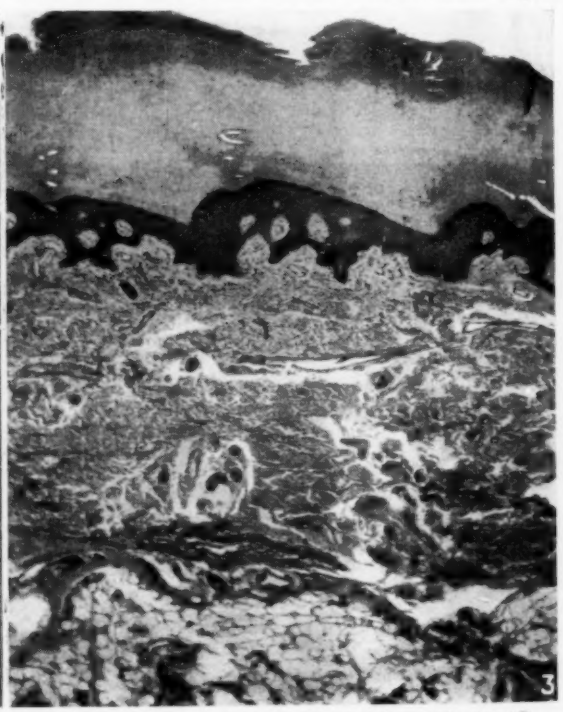

Fig. 3. Low-power section of normal palmar skin (× 45).

Fig. 4. This shows the bilateral lesions of tylosis on the palms.

lesions of the palms, and the office worker may have a predominant affection of the elbows.

The condition varies not only in intensity in different individuals of the same family but also at different times in an affected individual. The condition is aggravated during very warm or very cold weather and also during manual labour, especially if the work involves exposure of the hands to moisture and cold.[24] The influence of climatic and occupational factors was also well illustrated in members of the family we studied.

Many subjects may be so slightly affected that they do not realize that they have the condition until their attention is drawn to it. On the other hand, marked thickening of the palmar skin may reduce tactile sensibility and interfere with the finer finger movements. Pain is not usually a marked feature of the hand lesions, although Anderson[1] reported a case with extremely painful lesions of the hands. The soles tend to be more painful than the palms and this may interfere with normal walking. Affected areas are predisposed to fissuring because of a lack of normal elasticity.[22,23] The normal fissures are exaggerated and produce a mosaic-like appearance. Involvement of the dermis by the fissures causes pain and sometimes haemorrhage.

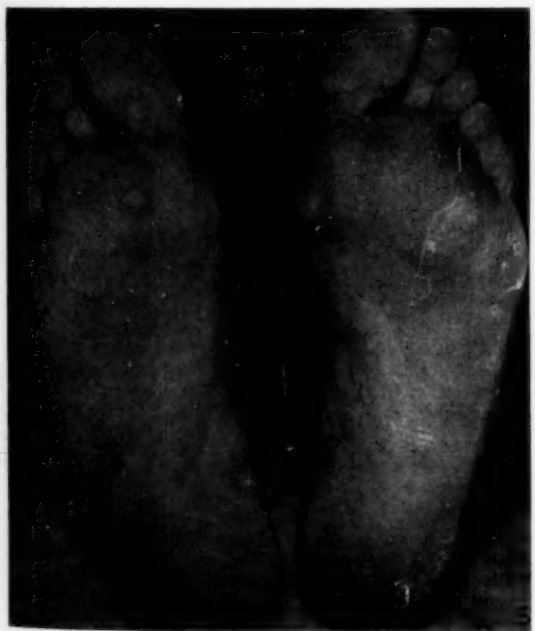

Fig. 5. This shows the bilateral lesions of tylosis on the soles. Note symmetry of the lesions.

Hyperhydrosis is present in the majority of cases reported in the literature. Families are on record in which sweating was diminished.[3] In the family under consideration both states obtained. There appeared to be an inverse relationship between the degree of hyperkeratosis and the amount of sweating, and we suggest that the hypohydrosis in severely affected cases may be due to obstruction of the sweat-gland ducts by the thickened and hyperkeratotic skin. Sweat-gland hypertrophy is not an uncommon feature and may account for the increased sweating noted in milder cases.

Tylosis should be differentiated from the acquired types of hyperkeratosis, notably lichen simplex (neurodermatitis), contact dermatitis, psoriasis, tertiary syphilis, fungal infections (particularly *Trichophyton rubrum*), volar verrucae, calluses, and the now rarely-seen arsenical keratoderma.[19,23,28]

Certain familial skin conditions resembling tylosis must be differentiated. Hereditary disseminate keratoderma palmaris et plantaris consists of multiple, symmetrical, discrete plaques on the palms and soles. The lesions do not coalesce and the onset usually occurs at adolescence but is sometimes delayed until adulthood.[21]

Mal de Meleda[3] is a rare type of palmar and plantar hyperkeratosis described only on the island of Meleda off the Dalmatian coast. Most of the inhabitants are consanguinously related; this favours transmission of the disease by what is considered to be a recessive gene. In addition to the usual sites the hyperkeratosis involves the dorsum of the hands and feet, and may extend up the legs and forearms to the elbows and knees. Hyperhydrosis is present.

There is no known cure for tylosis. Amelioration may occur with change of occupation or during humid weather. Treatment is purely palliative and numerous therapeutic measures have been employed with variable temporary benefit. Keratolytics such as salicylic-acid preparations are employed to soften and remove the horny layers. Superficial X-ray therapy has been advocated by some but has not met with general approval, mainly because of the risk of causing carcinoma and because effective dosage may lead to cicatricial atrophy with telangiectasis.[10] Various hormones, notably thyroid extract and oestrogens, have been tried. The use of large doses of vitamin A has been reported to control the gross manifestations of the disease.[16] Its use is purely empirical and no satisfactory explanation of the mechanism is forthcoming. Sandpapering and mechanical abrasion of the affected areas has been used. In severe cases benefit has been claimed by complete excision and grafting.[4,13,19,29]

Tylosis and Associated Abnormalities

Many abnormalities associated with tylosis have been

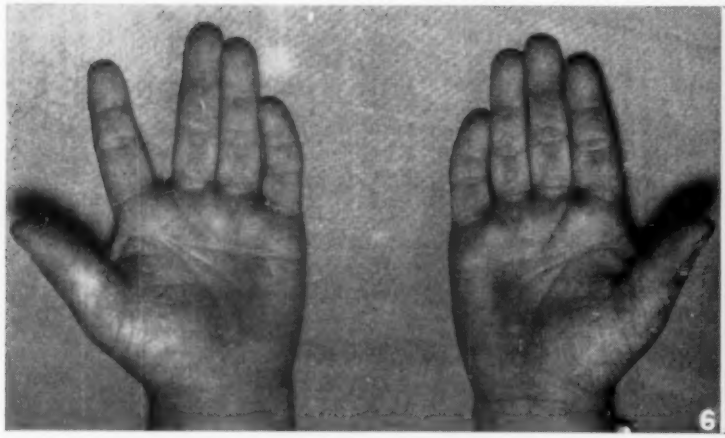

Fig. 6. Tylosis and clinodactyly. Note radial deflection of both little fingers.
Fig. 7. Clinodactyly. X-ray showing sloping of the distal surface of the middle phalanx of the little finger due to shortening of the radial surface of the phalanx.

described in isolated instances. Howel-Evans *et al.*[9] reported the association of carcinoma of the oesophagus and tylosis in 2 families. There was unequivocal evidence that carcinoma of the oesophagus was associated with tylosis in 17 of their cases, and in only 1 case in which the neoplasm was found was it impossible to establish that tylosis was present. Members of the family unaffected by tylosis were unaffected by carcinoma. In the cases of carcinoma of the oesophagus in which the oesophageal epithelium adjacent to the tumour was examined histologically, no evidence of hyperkeratosis was found. The association between palmar and plantar hyperkeratosis and leukoplakia of the mouth has been recorded.[12,23,26] Malignant degeneration in areas of hyperkeratosis has been considered likely by some dermatologists,[15] and Ingram and Brain[10] mentioned a case in which squamous carcinoma developed in tylosis.

Hanhart (quoted by Cockayne[3]) described a Swiss family in which the affected members developed tylosis and multiple lipomata during late adolescence. Cockayne[3] commented that the late age of appearance of tylosis made it doubtful whether it was the ordinary form of the disease.

Tylosis has been reported to occur occasionally with extensive ichthyosis hystrix or with epidermolysis bullosa.[10] It has been found associated with other ectodermal abnormalities[3,10,23] or with hypogenitalism, oxycephaly, clubbing of the fingers, or mental retardation.[2,3]

Clinodactyly, as described in our cases, is a shortening of the middle phalanx of the fifth finger, mainly on its radial aspect, which results in a radial deflection of the terminal phalanx of from 15° to 30° (Fig. 6). The remaining digits are normal and there is no flexion curvature.[8] On X-ray (Fig. 7), the middle phalanx of the little finger is slightly shorter on the radial side, thus forming an appreciable slope on its distal surface. X-ray observations indicate that this seems to be caused by a slowing-down process of ossification, specifically at the upper radial part of the middle phalanx of the fifth finger. Due to this slanting, the distal phalanx is inclined inwards towards the other fingers. The rest of the phalanges are normal. The incidence has been calculated at 1 in 1,000 in Northern Ohio.[8] Hersh *et al.*[8] feel that the term clinodactyly should be limited to those cases which are due to incomplete ossification and should not be confused with other causes of familial crooked little fingers such as those due to abnormal tendons[5-7] and to fused ossification.[11]

Genetic Transmission

Tylosis is inherited as a Mendelian dominant with high penetrance. It appears to be controlled by a single autosomal gene. Cockayne[3] reviewed 47 families in the literature and the proportion of affected to normal members was 594 : 483. The sex ratio was 318 males to 284 females. He reported 2 small families in whom the lesion only occurred in the females and not in the males. Subsequent studies, however, indicated that the two sexes are equally affected and no race is immune.[20,23]

Lawler and Renwick[14] are of the opinion that there are at least 2 types of inherited tylosis which apparently run true in families and appear to be due to different genes. The 2 forms have been differentiated on clinical grounds. Type A has a variable age of onset ranging from 5 to 15 years, whereas Type B is recognizable during the first year of life. The latter is clinically distinguishable from Type A by the uniform thickness of the keratosis, by the sharply delimited edges of the lesion, and by the rare incidence of painful fissuring. Both types are inherited as a Mendelian dominant. This clear-cut distinction was not a feature of our cases, since fissuring was not an uncommon finding in lesions which had been noted in the first year of life with relatively sharply demarcated edges and in which keratosis was of uniform thickness.

Tylosis is due to a mutant gene and the extent, distribution and severity are independent aspects which are largely determined by composite genetic and environmental factors. Thus the same mutant gene may cause several distinct clinicopathological pictures and this difference in expressivity may be evoked to explain the large and varied nomenclature applied to the hyperkeratoses.

Clinodactyly has not previously been described asso-

ciated with tylosis. The only reported association of clinodactyly is that with webbing of the toes.[8] In the family we studied there were 10 members with clinodactyly — 6 female and 4 male. In 8 cases the curving of the little finger was bilateral but in 2 this curving was only present in the left hand. Of those with clinodactyly, 6 were affected with tylosis as well. Clinodactyly is transmitted as an autosomal dominant. Lack of complete penetrance was shown by the fact that in 6 cases the parents did not have the condition. In 1 case (IV24) neither the parents nor the grandparents had clinodactyly. These features of dominant transmission with incomplete penetrance have been reported by others.[8]

Is there a genetic linkage between clinodactyly and tylosis, or is the association purely one of chance? We were unable to obtain statistically significant results in favour of genetic linkage, using the sibling pair method of Penrose.[17,18] However, assuming that the incidence of tylosis and clinodactyly in South Africa is 1 in 40,000[24] and 1 in 1,000[8] respectively, the probability of these 2 conditions occurring together is in the region of 1 in 40 million.

SUMMARY

In a family affected with tylosis palmaris et plantaris 59 members were studied. The family tree and the mode of genetic transmission are outlined. The incidence, histopathology, clinical features, differential diagnosis and treatment of tylosis are discussed. The association of tylosis and clinodactyly is noted. In addition some of the reported abnormalities occurring with tylosis are reviewed.

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OBSERVATIONS ON A NEW BENZOTHIADIAZINE DIURETIC—TRICHLORMETHIAZIDE*

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The introduction of the benzothiadiazine diuretics has had a marked influence on the treatment of patients with oedema. Modifications of the original molecule have resulted in preparations which, in the main, vary quantitatively in potency but not qualitatively. This paper presents the effects observed of a recently developed derivative, trichlormethiazide (3-dichloro-methyl-6-chloro-7-sulfamyl-3, 4-dihydro-1, 2, 4-benzothiadiazine-1, 1-dioxide) (Fig. 1), on urinary electrolyte excretion in normal subjects and on patients with fluid retention.

Normal Subjects

Three healthy male medical students were studied. They were placed on a standard hospital diet for 11 days. During the first 4 days (control period) the 24-hour excretion of water, sodium, potassium, chloride, bicarbonate, and the pH of the urine was measured as well as the weight of each subject. During the next 7 days, trichlormethiazide was given in doses of 4 mg. *b.d.* Three grams of potassium chloride were administered in divided doses daily during the last 3 days of this period. Measurements of weight, urine volume, and electrolyte content

TABLE I. EFFECT OF TREATMENT IN HEALTHY SUBJECTS*

	Subject 1			
		mEq		
Days	*Urinary vol. ml.*	*Na*	*K*	*Cl*
1 - 4 contro	1,345	164	85	187
5	2,800	338	986	380
6	1,810	236	131·9	240
7	1,770	159	126·6	206
8	800	65·4	75·5	88·5
9	940	71·5	110·1	83
10	1,670	210	266	266
11	1,960	160	115	225

* Effect of 4 mg. of trichlormethiazide twice daily on urinary volume, sodium, potassium and chloride excretion in 3 healthy subjects after a control period of 4 days. Three g. of potassium chloride were given daily for the last 3 days.

* Trichlormethiazide ('fluitran') was made available for study through Dr. M. Tonkin, Medical Director, Scherag (Pty.) Ltd.

** Final year medical students.

TABLE I. (CONTD.) EFFECT OF TREATMENT IN HEALTHY SUBJECTS

Days	Urinary vol. ml.	mEq Na	mEq K	mEq Cl
	Subject 2			
1 - 4 control	1,510	150	82	176
5	1,700	185	136	178
6	1,650	152	118	208
7	2,220	182	134	229
8	1,830	209	75	211
9	1,680	147	88·5	188
10	1,140	111	83	163
11	870	121	78	166
	Subject 3			
1 - 4 control	1,251	180	76	236
5	1,530	345·1	81·5	306
6	1,000	138·6	80·6	186
7	1,770	71·3	112·7	204
8	1,050	106·3	109·2	131
9	1,050	43·5	61	64·2
10	730	93·5	74·5	93·5
11	900	133	65	151

TABLE II. EFFECT OF TREATMENT IN HEALTHY SUBJECTS—(CONTD.)*

Days	Na : K Subjects 1	2	3	Na : Cl Subjects 1	2	3
1 - 4 control	1·92	1·83	2·36	·88	·88	·76
5	3·43	1·36	4·24	·89	·67	1·16
6	1·79	1·30	1·71	·99	·74	·73
7	1·26	1·36	·64	·78	·80	·35
8	·86	2·79	·98	·74	1·00	·81
9	·66	1·66	·72	·87	·78	·68
10	·79	1·33	1·26	·79	·68	1·00
11	1·15	1·55	2·04	·75	·73	·88

* Na] : K and Na Cl urinary excretion ratios before and during the administration of 4 mg. of trichlormethiazide twice daily to 3 healthy subjects.

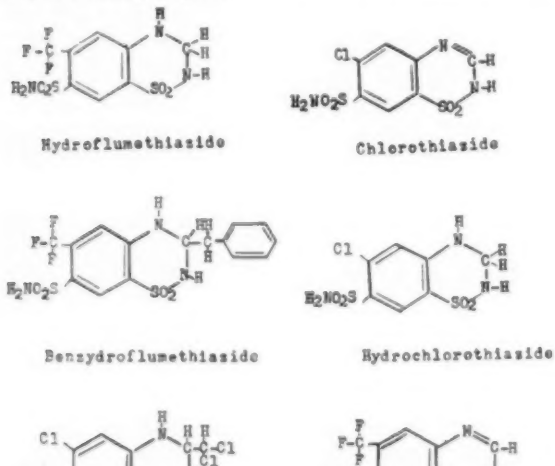

Fig. 1. The chemical formulae of some of the benzothiadiazine diuretics are shown. These are all substitutes at position 3 of chlorothiazide or flumethiazide.

were continued throughout the study, the urine being collected and preserved with toluene and thymol. The results are shown in Tables I and II and Fig. 2.

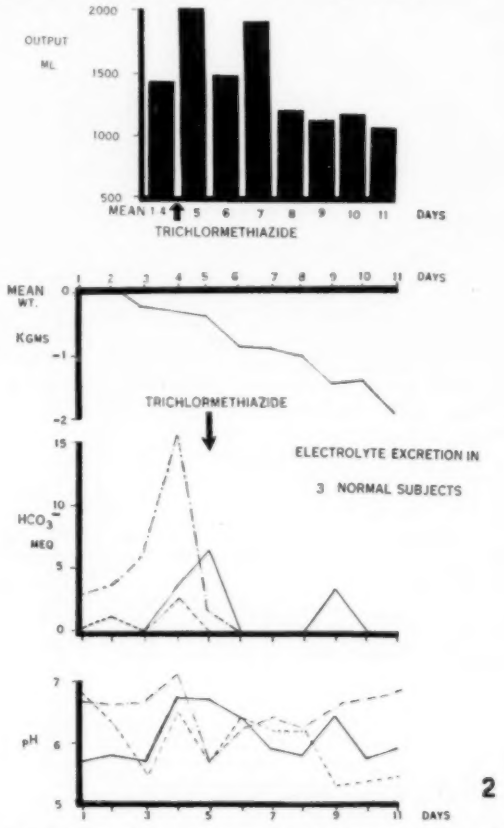

Fig. 2. The effect of 4 mg. of trichlormethiazide twice daily on body weight, bicarbonate excretion, and urinary volume is shown.

In 2 other healthy subjects the time of onset and duration of action of trichlormethiazide were studied. Urine was collected as above in periods from 8 a.m. - 2 p.m., 2 p.m. - 8 p.m. and 8 p.m. - 8 a.m. over 3 days while on a standard hospital diet. Trichlormethiazide (8 mg.) was then given and the effect on urine volume and electrolyte excretion observed for a further 60 hours. The results are shown in Figs. 3 and 4.

Patients with Fluid Retention

17 patients with fluid retention (9 with cardiac failure, 1 with the nephrotic syndrome, and 1 with steroid-induced oedema) were treated with 4 mg. of trichlormethiazide twice daily. Two patients had gross oedema, 9 mild oedema, and 6 had left ventricular failure without manifest oedema. Potassium supplements were not given during the 10 or 11 days of observation. In the patients with cardiac failure, specific treatment, excluding other diuretics, was given if indicated. Daily weight and frequent blood-electrolyte levels were recorded in each patient

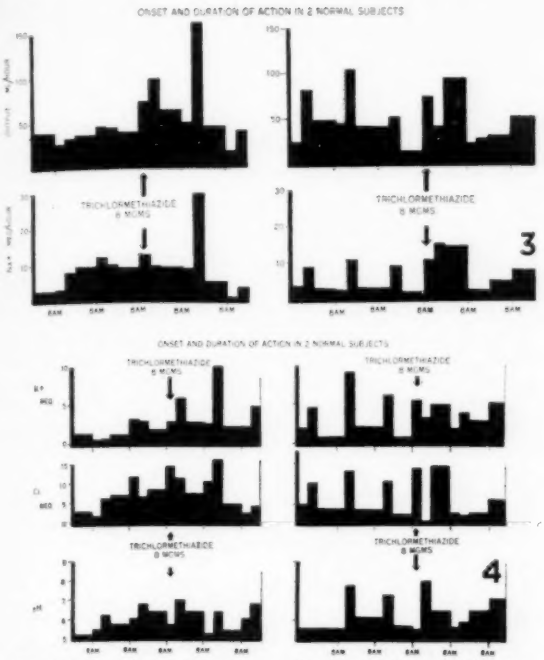

Fig. 3. The effect of an 8 mg. dose of trichlormethiazide on urinary output and sodium excretion in respect of its onset and duration of action is shown.

Fig. 4. The effect of an 8 mg. dose of trichlormethiazide on potassium, chloride, and urinary pH in respect of its onset and duration of action is shown.

during the course of treatment. The mean cumulative weight change and the mean serum-potassium change are shown in Fig. 5. There was no demonstrable alteration in the other blood electrolytes or urea. Table III shows the effects of trichlormethiazide on the urinary excretion of electrolytes in a patient with oedema.

TABLE III. EFFECT OF TREATMENT IN A PATIENT WITH OEDEMA*

		m.Eq.			
Days	*Urinary vol. ml.*	*Na*	*K*	*Cl*	*pH*
1 - 2 control	1,205	180	33	164	6·3
3	1,840	200	34·8	214	6·4
4	1,190	212	40·2	214	6·4
5	1,410	184	38·4	190	6·0
6	—	—	—	—	—
7	1,430	105	58·5	177	4·9
8	2,290	284	91·6	334	5·7
9	1,280	174	67·5	200	5·2
10	1,430	124	89·3	183	5·2

* Effect of 4 mg. of trichlormethiazide twice daily on urinary volume, sodium, potassium, chloride and pH in a patient with fluid retention.

DISCUSSION

The benzothiadiazines exert their effect, to a large extent, at the proximal convoluted tubule where they inhibit the reabsorption of a portion of the filtered sodium. This results in an osmotic diuresis. Moreover, there is an effect on the distal convoluted tubule of variable degree resulting

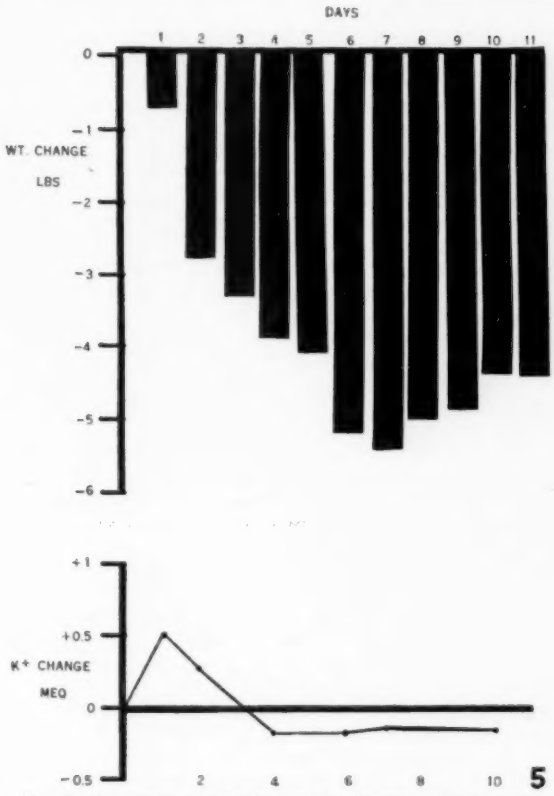

Fig. 5. The mean cumulative weight change and the mean serum potassium level is shown over 11 days of treatment with trichlormethiazide in 17 patients with fluid retention.

from the inhibition of carbonic anhydrase. The more recent derivatives have, however, little carbonic-anhydrase-inhibiting effect. As the molecule of chlorothiazide has been manipulated, so has the effective dose of the derivatives decreased and it has been suggested that this increased activity may be due to an increased solubility of the drug in the lipoid of the cell membrane.[3] A more specific interference with energy production in the cell is not, however, excluded, and possible differences in electrolyte excretion produced by the different preparations may result from this.

The administration of 4 mg. of trichlormethiazide twice daily to 3 normal subjects resulted in an increase in urine volume, sodium, and chloride excretion and, to a lesser extent, potassium. Urinary pH and bicarbonate excretion were not altered to any considerable extent. In 2 subjects the ratio of sodium to chloride excretion in the urine increased temporarily and then decreased when compared with control levels. In the same subjects similar changes were noted in the sodium: potassium ratio. The results are consistent with a primary action on sodium reabsorption and secondary osmotic diuresis. The absence of an increase in urine pH or bicarbonate excretion suggests little if any carbonic-anhydrase-inhibiting effect of the drug and is similar to that reported as occurring with

the 3-benzyl derivatives[2] and to a lesser extent with hydroflumethiazide.[3] The secondary retention of sodium observed after a few days suggests that a compensatory mechanism to restore body sodium began to exert an effect. This may be due to increased secretion of aldosterone as evidenced by a decrease in the sodium-potassium ratio and is to be expected in normal subjects depleted of water and sodium.[4] It is unlikely that other diuretics with similar modes of action would differ in this respect.

There was no marked increase in the absolute amount of potassium excreted, except in one subject in which a greater urinary excretion was seen to follow the administration of potassium supplements. Similar observations, that trichlormethiazide and bendroflumethiazide tend to cause less potassium loss than chlorothiazide, hydrochlorthiazide, flumethiazide and hydroflumethiazide, have been made by Bernstein.[5] A greater carbonic-anhydrase-inhibiting effect of the latter substances may account for this.

A single dose of 8 mg. of trichlormethiazide was found to exert an action within 6 hours of administration. The effect continued for 24 - 36 hours and was observed to abolish the normal diurnal rhythm of water and electrolyte excretion in the urine (subject 2, Figs. 2 and 3). It has also been stated that there is a continued water diuresis without electrolyte loss following cessation of continuous therapy of 7 days duration.[6] This has been associated with a decrease in serum osmolarity produced by the previous treatment. It would appear then that trichlormethiazide has a long duration of action and may be better given in intermittent courses with continued diuretic effect both during and for a period after stopping the drug. If administered in this manner, hyponatraemia, which presumably may develop on continuous therapy, would not occur.

Satisfactory diuresis was observed in the 17 patients treated with trichlormethiazide. There was a mean cumulative weight loss of 5½ lb. in these patients in 1 week. Comparisons with other diuretics cannot be made accurately with this heterogenous group of patients. The diuresis observed was satisfactory while the rate of clinical improvement was similar to the expected results with other diuretics, and Ford[7] has shown that 5 mg. of trichlormethiazide is equivalent to 2 ml. meralluride in natriuretic effect. In some patients the weight increased after a week, and was unassociated with signs of fluid retention. It is considered likely that this resulted from an increase in food intake and consequent increase in actual body solids. Although during the period of treatment there was no drop in the mean serum-potassium level, in some patients a small decrease was observed. Thus, although potassium depletion may be less likely to occur with this than with previous preparations, as judged by comparative urinary excretion, it may still be necessary to supplement potassium intake. During the period of observation no significant change in other blood electrolytes or the blood urea was observed, although in subsequent patients on high dosage a rise in blood urea has been observed in some. As with the other benzothiadiazine derivatives, it is likely that in patients with renal insufficiency, worsening of kidney function may occur with vigorous sodium depletion. Care should be exercised in these cases. Similarly, an increase in the serum uric-acid level may occur with trichlormethiazide as with any of the benzothiadiazines.

The LD_{50} dose of trichlormethiazide is given as 2·8 g./kg. in mice. Hydrochlorothiazide has a similar LD_{50} dose, but, because of the larger dose necessary to produce a diuretic effect, the therapeutic ratio is not as high as for trichlormethiazide.[8] This may be of advantage in that toxic effects would be less likely to occur with the smaller dose required when using trichlormethiazide. However, the therapeutic ratio is still considerable with the other benzothiadiazines and well within the toxic dose. Idiosyncrasies are independent of dose and may occur with either. In this series of subjects and in other patients treated concomitantly, no side-effects, toxic effects, or idiosyncrasies were observed.

SUMMARY AND CONCLUSIONS

The effects of trichlormethiazide, a benzothiadiazine derivative, were observed in 5 normal subjects and in 17 patients with fluid retention.

Increased excretion of sodium, chloride, water, and to a lesser extent potassium, was observed after administration of 4 mg. twice daily to normal subjects. The effect was measurable within 6 hours and persisted for 24 - 36 hours.

In the patients with fluid retention a satisfactory diuretic response was observed. Potassium depletion was slight and may be less than that reported for other benzothiadiazine derivatives.

No side-effects, toxic effects, or idiosyncrasies to the drug were noted.

We wish to thank Prof. G. A. Elliott for his encouragement and criticism, Messrs. V. Noble, M. Brooks and N. Weston for technical assistance, and the Photographic Unit of the Department of Medicine for the illustrations.

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THE SECOND WORLD CONFERENCE ON MEDICAL EDUCATION*

B. J. KAPLAN, M.B., B.Ch., *Senior Lecturer in Social Medicine, University of Cape Town*

The Second World Conference on Medical Education was held in Palmer House, Chicago, from 30 August to 4 September 1959. About 650 delegates attended from 66 countries, more than half the delegates being from countries outside the USA. Three South Africans were present, Mr. T. B. McMurray of Cape Town, Dr. B. Kaminer of Johannesburg, and myself. Delegates came from 65 of the 98 medical schools in the USA and and it is estimated that about 60,000 students graduate annually from the 555 medical schools which were represented. One example of the progress of medical education is the fact that there are now 67 medical schools in India.

* Received for publication on 13 January 1961.

Dr. Raymond B. Allen (California) was President of the Conference, which was under the patronage of President D. D. Eisenhower.

Tours of medical institutions in Chicago (including 5 medical schools) were arranged. There was a programme of medical films and a commercial exhibition. Social facilities included opportunities for meeting other delegates in the Palmer House hotel, an official banquet, and two receptions (cocktail parties). Many delegates were entertained in the homes of Chicago doctors, a special night having been set aside for this purpose.

The Conference had as its theme 'Medicine — a life-long study'. There were 2 days of plenary sessions, and on 3 days the conference met in 4 sections, namely:

1. Basic clinical training for all doctors.
2. Advanced training for general and speciality practice.
3. The development of teachers and investigators.
4. Continuing medical education.

It was thus impossible for any individual to attend all the proceedings of the conference and the picture presented here is coloured by my personal interests.

MEDICINE — A LIFE-LONG STUDY

It seemed to be generally agreed that the present medical curriculum is failing to stimulate students to pursue the acquisition of knowledge after graduation. Such stimulation can only be effective if the doctor has a knowledge of statistics and an appreciation of current medical literature. Many doctors, after graduation, lead isolated medical lives with little, if any, contact with hospitals. It was felt that these contacts with colleagues in the hospital as well as in other groups provided the essential means of a continuing interest in medical knowledge.

THE CURRICULUM

Many speakers were against the didactic method of teaching and against systematic lectures. 'Students may go into a lecture and out again without actually attending.' Frequently the medical student was too busy to think and too tired to read or experiment.

'In general, it may be said that the curricula in most medical schools are recognized to be grossly overloaded, unbalanced, and as showing no clear evidence of policy or design, but rather only of having grown up by a process of relatively incoordinated accretion ... Even the students' clinical work tends to be split up into a series of specialized and uncoordinated sections ... We should instruct less and educate more' (J. G. McCrie, Sheffield).

It is 'impossible to teach in medical school all that a physician should know'. The intensive specialization, which is such a prominent feature of modern medicine, has led to an overly technical approach to meet the rapid advances in medical knowledge. Too much emphasis has been placed on teaching the technical aspects of medical practice and too little on preparing the doctor for his traditional rôle of guide, philosopher, and friend. Students must be made to see the importance of the humanities and social sciences. In asking the following questions, J. H. F. Brotherston, however, struck a warning note: what would be the repercussions of drastic changes in the fundamental curriculum? To what extent is the international unity and identity of the medical profession due to the uniform training which we all receive? Careful planning of the curriculum to meet the aims of medical education is therefore essential.

Sir George Pickering felt that the teaching of method and the conveying of good habits were the chief aims of medical education: 'What is the difference between basic principles and prejudice?' There are special subjects and special techniques to be conveyed to the student. Sir George emphasized the student's need for familiarity with people in general and with the patients with whom he is going to work.

The student must be taught methods of solving unfamiliar problems and he should acquire habits of self-education and enthusiasm for renewing the study of medical science. He requires to have a sense of responsibility to his patients. He should be able to criticize his own efforts and to assess the value of what he hears from others, or reads in the literature. In other words, it is the task of the medical school to direct his development and the best way to achieve this seemed, in the opinion of many delegates to the conference, to be the promotion of individual effort by each student. 'Medicine is learned, not taught.'

THE STUDENT REMEMBERS WHAT HE DOES

Many curricula now require each student to write a paper which includes a survey of resources available and necessitates reference to the literature. It is realized that many students may never again write a paper and that this one effort may be an invaluable lesson in being conservative in coming to reasonable conclusions. These papers proved of value also to members of staff who were thus enabled to select suitable individuals for research work and for cultivation as future teachers.

Other ways of promoting student participation in their own education were:

1. Seminars of small groups of students. In small groups each student participates and remains on his toes.
2. Preparation of subjects for discussion with other members of the class.
3. Responsibility for the care of patients, with adequate supervision. Clinical training of students should be centered around a series of graduated stages of student participation and responsibility.
4. Practical classes with set problems to be solved, to illustrate different techniques which are available.

How Much Did the Student Learn

Considerable emphasis was placed on the difficulties in assessing the student's absorption of knowledge. Many said that the system of written examinations is unsatisfactory. It was indicated that this is a virgin field for research and it was, in fact, suggested as the theme for the next conference on medical education.

DIFFICULTIES CONNECTED WITH TEACHERS

Many difficulties in medical education arose from the fact that most medical teachers have had no pedagogic training. An excellent conference was recently held in Buffalo, and a report, edited by George E. Miller and E. F. Rosinski entitled 'A summer institute on medical teaching' appeared in the *Journal of Medical Education*, May 1959 (**34**, 449).

Research and teaching are inseparable. An important question is how to attract the gifted individual to teaching and research.

The example which a teacher sets to his students was considered of major importance. Contact of a small group of students with one teacher was felt to be of greater value than 'rapid floating from one member of staff to another'.

Visual Aids

I saw closed-circuit television in action on several occasions and it was estimated that the apparatus necessary for a self-contained unit would cost in the region of £3,000. Several catalogues of medical films have been deposited in the Medical Library of the University of Cape Town. The financial difficulties in either borrowing or purchasing films are, however, very considerable. Films vary in quality, and should be seen before money is spent on them.

THE CHANGING DEMANDS OF MEDICAL PRACTICE

The advance of medical knowledge and specialization, with its resultant emphasis on the technical aspects of medical treatment, is making an increasing number of doctors aware of the patient's need for personal services and for total, comprehensive medical care. Such comprehensive care demands the collaboration of a team of workers for the therapy of most patients. Such a team consists not only of a group of specialist doctors, but also of ancillary workers such as nurses, social workers, physiotherapists, and psychologists. In order to convey to the student the outlook which is necessary by a doctor who functions as part of the team, it was felt that collaboration and team-work should be emphasized in the curriculum.

Nobody spoke with satisfaction about the lot of the general practitioner, and there was disagreement about his functions. The training of a specialist was said to be technical

and easy, whereas the training of a family practitioner is difficult and unsatisfactory.

The training of general practitioners is now in the hands of specialists and it is not surprising that the good student finds the prospect of general practice distasteful. Few medical schools have given an adequate concept of general pactice to the student.

The majority of medical students, once they have completed their internship, enter general practice. Yet, the general practitioner had long ago been removed from membership of a medical faculty.

Brotherston (Edinburgh) submitted that a senior and competent general practitioner is a specialist in his own right. It was pointed out that the general practitioner usually has a knowledge of the cardiac, gynaecological, family, social, economic, and other circumstances of his patient. These many aspects, all essential to the patient's health, are not usually known by the specialist. The general practitioner is therefore probably the best equipped to guide and to treat the patient. I suggest that the general practitioner, or family doctor, should be the head or director of the team which is to provide comprehensive medical care.

There is a marked difference in the nature of illness between cases seen in hospital and elsewhere (outside). Brotherston compared the *'in vivo'* patient in his own clothing and domestic circumstances with the *'in vitro'* patient in hospital.

EXTRAMURAL TEACHING

More than 20 medical schools in the USA have adopted some form of teaching outside the hospital and many speakers emphasized this aspect. Many considered it essential for the student to have some knowledge of the patient's home and environment following personal experience. It is impossible to visualize a poverty-stricken home and its effects on the health of the patient, unless the student has had some experience of it. Students attend at outpatient clinics, health centres, or at special units. Many medical schools have now adapted family studies (including home visits and medical care in the home) as part of the curriculum. Some medical schools have used this family study as a research instrument in following up cases of particular diseases from the hospital. Several medical schools incorporate the utilization of non-medical resources as part of the curriculum: at Harvard students submit written reports on their survey of the resources available in a community for assisting patients, e.g. those that are aged, crippled, economically destitute, requiring rehabilitation, and cases of infectious disease needing isolation.

An increasing number of medical schools now have a preceptorship programme, whereby students volunteer for attachment to a general practitioner for a variable period of time. The student will 'sit in' with the general practitioner during his consulting hours and also go round on visits to homes, hospitals etc. Since October 1959 such a preceptorship is compulsory at the University of Edinburgh.

VACATION WORK

Several medical schools are providing facilities for students to assist with research work during the vacation. The value of such effort is enormously enhanced if the student writes up his findings. The Californian health authorities accept 30 students as observers during vacations and a grant is paid to students to help cover their cost of board and lodging.

INTERNSHIP IN PUBLIC HEALTH

A. M. Davies reported that a 13th month of compulsory internship had been introduced in Israel. During this month the young graduand was required to submit a written report, and a certificate of 'due performance' was necessary. Three students had to repeat their reports in 1957, but 30% of the reports were of a very high standard. One led to new legislation on conditions in industry. A student who was uninterested in preventive medicine had been instructed to follow up 50 patients who had been in the wards of the hospital. It had been hoped that this feature would lead to greater interest, but only one graduand had thus far entered the public health service.

Dr. R. M. Tadic (Yugoslavia) asked for a resolution in favour of such compulsory internship.

INTERNATIONAL EXCHANGE

The benefits of international travel were well recognized and encouraged by the conference. Dr. Moore reminded us that a travelling fellowship was not only a scientific pursuit, but the Fellow should see and participate in the (new) culture of the country which he was visiting.

CONCLUSION

The proceedings of the Second World Conference on Medical Education will be published during 1961. Together with the report (1954) of the First Conference which was held in London in 1953, a wealth of authoritative information, and debate by experts on controversial issues, will be available to anyone who has the progress of medicine or the status of its doctors at heart.

I am grateful to the University of Cape Town for special leave and to the International Epidemiological Association whose grant to attend another conference made my visit to Chicago practicable.

AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENT

TARIEF VIR GOEDGEKEURDE MEDIESE HULPVERENIGINGS

Kopieë van die Tariefboek in die desimale muntstelsel is aan al die lede van die Vereniging gepos. Indien enige lid sy eksemplaar aan die end van Februarie 1961 nog nie ontvang het nie, moet hy die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, daarvan in kennis stel.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
2 Februarie 1961

TARIFF OF FEES FOR APPROVED MEDICAL AID SOCIETIES

Copies of the Tariff Book in the decimal monetary system have been posted to every member of the Association. If any member has not received his copy by the end of February 1961 he should advise the Secretary, Medical Association, P.O. Box 643, Cape Town, of the fact.

L. M. Marchand
Associate Secretary

28 Plaza Building
Pretoria
2 February 1961

HART-LONG GROEP VAN DIE UNIVERSITEIT VAN STELLENBOSCH

'n Vergadering, bygewoon deur lede wat verteenwoordigend is van alle departemente met belangstelling in kardiopulmonale werk, is gehou op 9 Februarie 1961.

Dr. F. P. Retief het die geval van 'n getroude dame van 27 jaar met tipiese familiehipercholesterolemiese xantomatose voorgedra. Haar bloedcholesterol was 668 mg. per 100 ml. en xantelasma, xantomata tuberosum en tendinosum met die kliniese tekens van aortastenose, sowel as 'n geskiedenis wat isgemie van die miokard aandui, was teenwoordig. Verskeie familielede het soortgelyke velletsels, en 'n broer is op 30 jarige ouderdom ‚aan sy hart' oorlede.

'n Vereenvoudigde klassifikasie van die lipoïdoses is voorgestel (sien onder) en familiehipercholesterolemie in besonder is verder bespreek. Dit word as ‚onvolkome' dominant oorgeërf. Volgens sommige veroorsaak die heterosigotiese staat 'n ‚forme fruste' met slegs verhoogde cholesterol-waardes. Die tipiese sindroom dui op 'n homosigotiese toestand. Die

patogenese, hoewel nog onduidelik, word aan buitensporige cholesterol-vervaardiging as gevolg van aangebore ensiemafwykings toegeskryf. Die histologiese en kliniese beeld is bespreek met klem op ernstige kardiovaskulêre aantasting. Bogenoemde pasiënt het ook klagtes gehad wat galblaaspatologie aandui. Die cholesistogram was egter negatief. Hierdie komplikasie, hoewel teoreties te wagte, kom skynbaar selde voor.

KLASSIFIKASIE VAN DIE LIPOÏDOSES

Lipoïdose

1. *Primêre Xantomatose (Plasma Helder)*
(A) Hipercholesterolemiese xantomatose.
(*i*) Familiehipercholesterolemiese xantomatose
(*ii*) Sekondêre xantomatose. (*a*) miksedeem, en (*b*) chroniese galobstruksie.
(B) *Normocholesterolemiese xantomatose.*
(*i*) Hand-Schüller-Christian sindroom.
(*ii*) Eosinofiel-granuloom.
(*iii*) Letterer-Siwe se siekte.
(*iv*) Infiltrasie van tumore, chroniese inflammatoriese letsels, ens.

2. *Hiperlipemiese xantomatose* met sekondêre hipercholesterolemie (plasma troebel).
(*a*) Familie-hiperlipemiese xantomatose
(*b*) Sekondêre hiperlipemiese xantomatose: (*i*) Ernstige diabetes, (*ii*) chroniese pankreatitis, (*iii*) nefrose, (*iv*) Gaucher se siekte, en (*v*) Von Gierke se siekte.

3. *Ander lipoïdoses:* Gaucher se siekte, Niemann-Piek se siekte, en Tay-Sachs se siekte.

Dr. Louis Potgieter het hierna 'n beknopte oorsig van die huidige terapeutiese uitrusting in gevalle met hipercholesterolemie gegee.

In die daaropvolgende bespreking het prof. H. W. Weber met 'n patologiese monster die oorsaak van dood van 'n 8-jarige kind met xantoma tuberosum geïllustreer, waar 'n kroonaar deur 'n xantoom-neerslag afgesluit is. In antwoord op 'n vraag van dr. W. H. Opie aangaande die wenslikheid van insluiting van Groep 3 onder xantomatose, vereenselwig prof. Weber hom met die klassifikasie omdat vanuit patologiese oogpunt die toestande almal histiosiete is met abnormale lipoïed-versamelings, ongeag of dit cholesterol of 'n ander lipoïed is.

Dr. B. J. v. R. Dreyer het hierna 'n geval waarin chylotoraks as 'n komplikasie van 'n operasie vir ope ductus arteriosus voorgekom het, bespreek. Dit is die eerste geval met hierdie komplikasie wat bekend is na operasie vir ope ductus arteriosus.

'n Opsomming van die beskrewe nie-traumatiese en traumatiese oorsake van chylotoraks is gegee. Daar word gewys op die seldsaamheid van infeksie selfs met herhaalde aspirasie vanweë die hoë vetsuur-gehalte van die limfvog. Daar word ook op gewys dat waar nie-traumatiese oorsake 'n swak prognose dra, die traumatiese gevalle dikwels spontaan opklaar na aspirasie en dikwels nie verdere ingrepe nodig het nie. Dit was ook in hierdie geval nie nodig vir verdere chirurgie nie. Die toestand verskyn nie onmiddellik na 'n operasie nie omdat uithongering dan nie veel limf vir vervoer daarstel nie.

'n Interessante en genoeglike aand is afgesluit met verversings deur mevv. drs. Botha en De Kock.

AMERICAN RHINOLOGIC SOCIETY

AMERICAN SURGEONS TO PRESENT NASAL SURGERY POSTGRADUATE COURSE IN JERUSALEM

A group of 25 American specialists in nasal surgery will serve as instructors at the second International Postgraduate Course in 'Reconstructive surgery of the nasal septum and external pyramid', to be presented in Jerusalem, Israel, 6 - 17 August 1961.

The course will be under the auspices of the Department of Otorhinolaryngology, Mayer de Rothschild Hadassah University Hospital, and the Hebrew University-Hadassah Medical School of Jerusalem, in cooperation with the American Rhinologic Society. Lectures and demonstrations will be in English.

Dr. Maurice H. Cottle of Chicago, Professor of Otorhinolaryngology, Chicago Medical School, and founder of the American Rhinologic Society, will direct the course.

Contributions of friends of the American Rhinologic Society have made it possible to offer many tuition scholarships.

Further information may be obtained from Dr. Robert M. Hansen, Secretary, American Rhinologic Society, 2210 Lloyd Center, Portland 12, Oregon, USA, or to Dr. Moshe Prywes, Dean of the Hebrew University-Hadassah Medical School, Jerusalem, Israel.

REMOVAL OF CARDIO-RESPIRATORY ORGANS*

Cases in which medical practitioners have apparently neglected to carry out the statutory functions imposed on them by Section 29(2) of the Pneumoconiosis Act No. 57 of 1956 have, in recent months, been brought to the notice of the Bureau.

Omission by a medical practitioner to carry out his obligations as prescribed in the above Section of the Act, apart from being an offence punishable on conviction with a fine not exceeding fifty pounds, may have serious financial consequences for the dependants of the deceased person.

They may thereby be deprived of the financial benefits (which may include a monthly pension) for which provision is made in the Act.

In their own interest and in order to obviate recrimination at a later stage by dependants of deceased miners, medical practitioners who attend such persons at the time of their death should ascertain (from the dependants) whether they have at any time worked in a dusty atmosphere at controlled mines, and should at the same time endeavour to obtain written permission to remove the cardio-respiratory organs.

In this connection attention is invited to Bureau Circular No. 1/1958 which prescribes *inter alia*, the manner in which these organs should be removed, preserved, and dispatched to the Bureau. Copies of the circular may be obtained from the Pneumoconiosis Bureau, De Korte Street, Johannesburg.

* Circular No. 1 of 1961, Section 29 of the Pneumoconiosis Act 57 of 1956.

IN DIE VERBYGAAN : PASSING EVENTS

National Cancer Association of South Africa, Postgraduate Study Grants Relating to Cancer. The National Cancer Association announces that a *limited sum of money* is available during the calendar year 1961 for registered medical practitioners who wish to pursue a course of postgraduate study in the field of cancer. Preference will be given to applications related to the subject of *exfoliative cytology.*

Application forms and fuller particulars may be obtained from the National Secretary, P.O. Box 2000, Johannesburg. The closing date for receipt of applications will be 15 April 1961.

* * *

University of the Witwatersrand Medical Graduates Association: Refresher Course in Cardiology. An intensive weekend

Refresher Course in Cardiology is being organized with the assistance of the Department of Medicine's Cardiac Clinic and the Transvaal Cardiac Society. This will take place on the weekend of 14, 15 and 16 April, and will begin with an evening session on Friday, 14 April 1961.

The emphasis in this course will be to acquaint the practitioner with the recent developments in cardiology and will also provide him with the opportunity to further his own practical experience in this subject. Particular attention will be given, where possible, to practical case demonstrations.

The attendance at this course will, for obvious reasons, be strictly limited, and those interested are urged to apply as soon as possible to the Secretary, Medical Graduates Association, Medical School, Johannesburg. The fee is R6, payable with the application.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 13 March at 5.10 p.m. in the Institute Lecture Theatre. Dr. L. J. A. Loewenthal will speak on 'The value of laboratory services to the dermatologist'.

* * *

Department of Anaesthesia, University of Cape Town. On Saturday 11 March a meeting will be held in the A-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, at 9.30 a.m. Dr. M. J. Rorke will speak on 'Cardiac arrest'. All interested doctors are invited to attend this meeting.

* * *

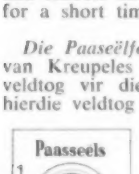
University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 8 March at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr. P. M. Smythe will speak on 'Tetanus'. All members of the Medical Association are welcome to attend this lecture.

* * *

Easter Stamp Fund. The National Council for the Care of Cripples in South Africa is again organizing the sale of Easter stamps. The proceeds of this annual effort enable the National Council and its 9 Cripple Care Associations throughout the Union and South West Africa to continue their work among cripples. They are striving to maintain and expand existing voluntary 'cripple-care' services. Practitioners engaged in the preventive, curative and rehabilitative fields of cripple-care, recognize that the work of these voluntary organizations is an essential part of the national campaign to prevent crippling. The important work of the associations can only be continued if they receive adequate financial support from the public.

The Council's sole source of income is derived from its Easter Stamp Fund. The campaign is launched annually in March, and the stamps will be on sale at most Post Offices and schools and outside large business houses from 1 March for a short time.

Die Paaseëlfonds. Die Nasionale Raad vir die Versorging van Kreupeles in Suid-Afrika hou weer eens hul jaarlikse veldtog vir die verkoop van Paasseëls. Die opbrengs van hierdie veldtog stel die Nasionale Raad en sy 9 geaffilieërde kreupelsorgverenigings dwarsdeur die Unie en Suidwes-Afrika in staat om hul werk onder kreupeles voort te sit. Hul strewe is om die bestaande vrywillige kreupelsorgdienste te onderhou en uit te brei. Praktisyne wat by die voorkoming, genesing en rehabilitasie van kreupeles betrokke is, erken dat hierdie vrywillige verenigings 'n essensiële rol speel in die nasionale veldtog om gebreklikheid te voorkom.



Die Paasseëlfonds is die Raad se enigste bron van inkomste en die verenigings kan hul belangrike werk alleen voortsit met behulp van die finansiële ondersteuning wat hulle van die publiek ontvang. Die veldtog word jaarliks in Maart gehou en seëls is vanaf 1 Maart by die meeste poskantore, skole en groot sakehuise te koop.

* * *

South African National Tuberculosis Association, Prophylactic Isoniazid Halts Tuberculous Infection among Nurses. For many years Dr. B. A. Dormer, Superintendent of the King George V Hospital in Durban and Chief Adviser on Tuberculosis to the Union Government, has been experimenting with the prophylactic use of the drug isoniazid among the hospital's nursing staff, and his findings are reported in the *Lancet* of 15 October 1960.

In September 1955 prophylaxis with isoniazid, 300 mg. daily, was initiated among the non-European nurse-aides in this tuberculosis hospital. These numbered about 600, of whom 16% were tuberculin-negative on appointment. In the next 2 years no case of tuberculosis was diagnosed in this group; in the third year only 1 case was diagnosed; but disappointingly in the fourth year 3 cases were diagnosed. Of these 4 cases arising after the introduction of isoniazid prophylaxis, 3 were detected while the nurse was on leave or within a month of her return from leave, thus indicating that she had neglected to take the drug while she was away. Subsequently supervision and propaganda have been intensified and nurses are urged to take isoniazid daily and to continue taking the drug while on leave and also for 3 months after leaving the staff.

The nurses who developed tuberculosis after a period of isoniazid prophylaxis responded very satisfactorily to treatment with isoniazid or its derivatives and streptomycin with or without PAS. The outcome, judged by the speed with which sputum was converted, X-rays cleared, and the nurses returned to duty, compares favourably with the outcome of cases in which tuberculosis developed without previous isoniazid prophylaxis. A recent questionnaire revealed that 32% of the nurses were taking isoniazid irregularly and 68% regularly.

* * *

South African National Tuberculosis Association, Official Opening, Doris Goodwin SANTA Centre. The Doris Goodwin SANTA Centre at Edendale, Pietermaritzburg, was officially opened on Monday 20 February 1961, by the Secretary for Health, Dr. B. M. Clark. This Centre admitted its first patients on 25 July 1960. It has 120 beds for Bantu TB patients and is now filled to capacity. The Centre is named after Councillor Mrs. D. E. Goodwin, Chairman of the Pietermaritzburg Branch of the Natal Anti-tuberculosis Association, who has been active in the campaign to control tuberculosis in the area for many years.

* * *

The Israel Medical Association cordially invites all its members and friends abroad to participate in the forthcoming 5th World Assembly of the Israel Medical Association to be held in Jerusalem, Haifa, and Tel-Aviv on 14 - 25 August 1961. Further information may be obtained from Dr. V. Resnekov, 102 Beverley Heights, Killarney, Johannesburg.

* * *

The Swedish National Association against Heart and Chest Disease, will award a Scholarship in 1961 of 25,000 Swedish crowns for 1 year, which should also cover travelling expenses.

The candidate, who should have proved ability in research, should be engaged in actual research in the field of cardiovascular disease and should be interested in studying a particular problem in Sweden. He should not be more than 45 years of age.

The applicants are expected to present a brief *curriculum vitae* with emphasis on research activities, together with details of proposed study in Sweden. Reprints of original works should be included. They should also be endorsed by their institutes (or clinics), which should ensure a post and research facilities on return.

Applications should be sent to: Chief, Cardiovascular Diseases Unit, World Health Organization, Palais des Nations, Geneva, Switzerland, before the end of March 1961. It is expected that the scholarship will be awarded in June 1961 and it is hoped that the candidate selected will be able to start his study in September 1961.

HAZARDS OF LIFE

The spectre of the want that might result from sickness or accident must haunt most men who rely on their own efforts for the care of their families and themselves. Elsewhere in this issue reference is made to a Personal Accident and Sickness Policy which the Association's Insurance Agency recommends for the consideration of members. The benefits are greater than those usually offered and the cover is wider.

It must be understood that this offer is not made to supersede the cover provided by the Professional Provident Society of South Africa which is non-cancellable, but is complementary to it and is specially designed for the professional man. To our knowledge, some of our members who have been in possession of one of these policies have benefited considerably following sickness or accident — a time when worry would have aggravated their condition and retarded convalescence.

It will be worthwhile to consider this offer which is only briefly outlined in the advertisement. The feeling of security and freedom from the fear of want, to be obtained by this means, is well worth the cost.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

SORBOQUEL

White Laboratories Inc., Kenilworth, USA, announce the introduction of Sorboquel, for the symptomatic control of the dual problem of acute and chronic diarrhoea — too fluid faeces, too rapid evacuations — and supply the following information:

Description. Each tablet contains 0·5 g. polycarbophil and 15 mg. thihexinol methylbromide.

Indications. Sorboquel tablets are indicated for the dual problem of acute and chronic diarrhoea — too fluid faeces, too frequent evacuations. Sorboquel is also indicated in the treatment of irritable bowel syndrome, regional enteritis, diverticulitis and ulcerative colitis, post-antibiotic enteritis, malabsorption syndrome, radiation proctitis, and surgically shortcircuited intestinal states.

Advantages. Polycarbophil is an inert synthesized macromolecular substance with an extraordinary water-binding capacity and is not absorbed from the intestine. Its marked absorption of free faecal water, characterizing diarrhoeal states, produces formed stools of normal consistency. The exceptional hydrosorptive action of polycarbophil is initiated only on reaching the slightly acid or alkaline medium of the small intestine or colon.

Dosage. Adults and older children: Initially 2 Sorboquel tablets followed by 1 *q.i.d.* is usually adequate. Depending on the severity of diarrhoea, divided daily dosage of 6 or even 8 tablets may be required initially in some cases. Doses exceeding 6 tablets a day should not be employed over prolonged periods. Maintenance dosage, 1 - 3 tablets daily.

Further information may be obtained from Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

PARNATE TABLETS

SKF Laboratories (Pty.) Ltd. announce the introduction of Parnate tablets and supply the following information:

Each Parnate tablet contains 10 mg. of trans-dl-2-phenylcyclopropylamine as the sulphate.

Indications. Parnate tablets are indicated for the treatment of pure depression (reactive, endogenous), involutional melancholia, manic-depressive psychosis (depressive phase), and psychotic depressive reactions.

Pharmacologically, Parnate is a mono-amine oxidase inhibitor with marked antidepressant properties. It is indicated in all types of depression, and has a considerably faster action than previous antidepressant agents. As it is not a hydrazine derivative it is free from the toxicity potential of the MAO inhibitors related to hydrazine. Maximum therapeutic benefit is often obtained within 7 days. There are no serious side-effects, and the action of the drug persists for only 48 - 72 hours after withdrawal.

Dosage. 10 mg. twice daily (morning and afternoon).

Presentation. Parnate is supplied in tablet form in containers of 25 and 250.

Further information may be obtained from SKF Laboratories (Pty.) Ltd., P.O. Box 38, Isando, Transvaal.

WESTPROD CARDIAC PACEMAKER

Westdene Products (Pty.) Ltd., announce the introduction of the Westprod Cardiac Pacemaker, manufactured by E. J. Middleton (Electronics) (Pty.) Ltd. of Johannesburg, and supply the following information:

This unit is a fully transistorized, battery/mains-operated pacemaker. Battery operation comes into effect immediately in the event of a power failure, or when disconnected from mains power to transport the patient from the operating theatre to the ward. There is a built-in switch for checking battery voltage. The rate of stimuli ranges from 35 to 180 per minute and is selective. An indicator bulb on the panel flashes with each stimulus. The voltage output to the heart

is adjustable from 0 to 20 volts. The voltage selector is a calibrated dial.

The built-in dual-purpose meter is a novel feature used for checking the battery voltage and indicating the actual current passed to the heart. Its usefulness cannot be underestimated when considering the fact that after prolonged use the electrodes, or 'heart wires' attached to the heart become covered with scar tissue. This factor causes less current to pass to the heart. The meter indicates this decrease. The operator, by applying more output voltage, can then return the voltage to its original intensity.

The Pacemaker is housed in a green metal cabinet, size 9×6×7 in., weighs only 5 lb. and is completely portable. Two insulated cardiac wires fitted with needles are supplied with each unit complete with electrode wires (not illustrated).

Further information may be obtained from Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

PARSTELIN TABLETS

SKF Laboratories (Pty.) Ltd. announce the introduction of Parstelin Tablets and supply the following information:

Each Parstelin tablet contains 10 mg. Parnate and 1 mg. Stelazine.

Indications. Parstelin is indicated in the treatment of: (1) Symptoms of depression and anxiety appearing concurrently; (2) emotional fatigue; (3) menopausal syndrome; and (4) emotional states, secondary to organic illness, and psychosomatic illnesses with symptoms of depression and anxiety.

Parstelin enables the physician to control *with a single preparation* the symptoms of depression and anxiety when they appear concurrently. The need for such a product has long been realized. The association of Parnate, a new anti-

depressant agent that is particularly effective in mild and moderate depressions, with Stelazine, a phenothiazine tranquillizer outstandingly effective in treating anxiety, is a logical development in this field.

Dosage. The usual dosage is 1 tablet twice a day (morning and afternoon).

Presentation. Parnate is supplied in tablet form in containers of 25 and 250.

Further information may be obtained from SKF Laboratories (Pty.) Ltd., P.O. Box 38, Isando, Transvaal.

INTRAVAL SODIUM

Maybaker (S.A.) (Pty.) Ltd. announce a change in the packing of Intraval sodium in that the pack of 6×0·5-g. ampoules with 6×10-ml. ampoules water for injection (for the preparation of the 5% solution) has been replaced by a pack of 5×0·5 g.-ampoules with 5×10-ml. water for injection.

Further information may be obtained from Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

SUPER HYDRAMIN POWDER

Nion Corporation, Los Angeles, California, announce the introduction of Super Hydramin Powder, and supply the following information:

Super Hydramin Powder is a powdered preparation of reduced volume providing 60% water-soluble high protein that is wholly assimilable. It is composed of proper proportions of all 10 essential amino-acids, plus 9 non-essential amino-acids, plus 18 associated vitamins and minerals combined with the essential minimum of fat and carbohydrate to prevent the possibility of constipation. Its content of all known dietary essentials, when mixed with the indicated amount of fluid, provides a balanced nourishment even for prolonged periods, either as the only source of food or as a supplement to diet.

Super Hydramin Powder avoids distension and contains no sugar or caloric sweetener. Its delicious taste is acceptable and enjoyed even by the hardest-to-please patients of all ages. One-quarter of a cup or ¼ oz. (2·123 g.) of Super Hydramin Powder contains 4·98 g. of carbohydrates, 0·05 g. of fat, 75 calories, and supplies 12·65 g. of the most recently improved pre-digested protein which is completely utilized by the body, and includes the following essential amino-acids: arginine, histidine, isoleucine, leucine, lysine, valine, methionine, phenyl alanine, threonine, and tryptophane. It also includes the following non-essential amino-acids: alanine, aspartic acid, cystine, glutamic acid, glycine, hydroxy proline, serine, tyrosine, and proline. Super Hydramin Powder also contains many vitamins and minerals.

Indications

In the case of underweight individuals of all ages, the dramatic change due to a restored amino-acid balance is often quite obvious with better blood values, better bone density, healthier condition of the skin and soft tissues, improved faecal elimination, etc.

Patients especially benefited by Super Hydramin Powder include: growing children, gastro-intestinal or thyroid surgery patients, persons with hepatic impairment, inoperable cancer, nitrogen loss from burns of fractures, or with ulcers or wounds that are slow to heal, cases of medical and surgical convalescence, in geriatrics, and weakened conditions caused by lingering illness, etc.

Super Hydramin Powder has also been tested and found ideal for weight control since it provides sufficient protein to replace the amount lost by daily metabolism and enables the patient to burn his own fat. It is an ideal invalid diet and for pregnancy and postnatal treatment; it supplies the extra demand for protein, vitamins and calcium.

Super Hydramin Powder is supplied in 16 oz. cans in a chocolate flavour.

Further information may be obtained from Vernleigh Products (Pty.) Ltd., P.O. Box 9027, Johannesburg, the distributors.

DORMWELL

Evans Medical announce the introduction of Dormwell Tablets and Dormwell Paediatric Tablets, manufactured by Smith & Nephew (Pharmaceuticals) Ltd., Welwyn Garden City, Herts., England, and supply the following information:

Dormwell is dichloralphenazone, a molecular complex of chloral hydrate and phenazone. It is a safe, non-barbiturate sedative and hypnotic.

Dormwell Tablets, available in containers of 25 and 250 tablets, each contain 10 gr. of dichloralphenazone; Dormwell Paediatric Tablets each contain 2½ gr.

As a hypnotic, dichloralphenazone induces drowsiness in 15 minutes and quiet, deep sleep within the hour, lasting for 5 - 8 hours. All the hypnotic and sedative properties of chloral hydrate are retained without the penetrating, unpleasant odour and the danger of gastric irritation. In addition, Dormwell possesses mild, analgesic properties which are an advantage in the management of insomnia associated with slight pain.

Dormwell Tablets are particularly suitable for elderly patients since they cause no restlessness or mental confusion. The paediatric tablets are very well tolerated by children and babies.

Dosage and Administration

Dormwell Tablets should be swallowed whole with a good draught of water.

Hypnotic: 2 tablets 20 minutes before retiring. Patients with a history of long and heavy sedation may need larger commencing doses.

Sedative: ½ - 2 tablets.

Dormwell Paediatric Tablets should be given in the following doses:

Hypnotic. Children 1 - 3 years: 1 - 2 tablets (maximum 4 tablets); 3 - 6 years: 2 - 3 tablets (maximum 6 tablets).

Sedative. Half the average hypnotic dose.

Literature and samples may be obtained from Evans Medical, P.O. Box 6607, Johannesburg.

BOEKE ONTVANG : BOOKS RECEIVED

The Handling of Chromosomes. 3rd revised edition. By C. D. Darlington, F.R.S. and L. F. La Cour. Pp. 248. Illustrations. 30s. net. London: George Allen & Unwin Ltd. South African Agents: Howard B. Timmins, P. O. Box 94, Cape Town. 1960.

Guide to Hygiene and Sanitation in Aviation. WHO Expert Committee on Hygiene and Sanitation in Aviation. Pp. 51. 18 figures. 3s. 6d. Geneva: World Health Organization. South African agents: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. 1960.

Official Records of the World Health Organization. No. 98. Pp. x + 283. 10s. 0d. Available also in French and Spanish. Geneva: World Health Organization. South African agents: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. 1959.

Annual Epidemiological and Vital Statistics, 1956. Pp. 705. £3 0s. 0d. Bilingual edition (French and English). Geneva: World Health Organization. South African agents: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. 1959.

Pathology of Infancy and Childhood. By Agnes R. Macgregor, M.D., F.R.C.P.E., F.R.C.O.G. Pp. viii + 631. 387 illustrations. 75s. plus 2s. 7d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1960.

The Ego in Love and Sexuality. By Edrita Fried, Ph.D. Pp. 296. $5·50. London and New York: Grune & Stratton, Inc. 1960.

Psychiatry: Descriptive and Dynamic. By J. A. Smith, M.D., F.A.C.P. Pp. 342. 56s. 0d. London: Baillière, Tindall & Cox Ltd. 1960.

Some Papers on the Cerebral Cortex. Translated from the French and German by Gerhardt von Bonin. Pp. xxiv + 396. 42 figures. 92s. 0d. Springfield: Charles C. Thomas. Oxford: Blackwell Scientific Publications Ltd. 1960.

BOEKBESPREKINGS : BOOK REVIEWS

ANAESTHESIA FOR INFANTS AND CHILDREN

Anesthesia for Infants and Children. By Robert M. Smith, M.D. Pp. 418. 182 illustrations. South African price: £5 2s. 0d. Local agents: P. B. Mayer, P.O. Box 713, Cape Town, and Westdene Products (Pty.) Ltd. Medical Book Department, P.O. Box 7710, Johannesburg. St. Louis: C. V. Mosby Co. 1959.

'It has been suggested repeatedly that paediatric anaesthesia carries a disproportionately high mortality. Statistical reports . . . show anaesthetic deaths to be higher in the first decade than in any other until the seventh decade is reached. Similarly, a survey on cardiac arrest reported by Stephenson and Reid points to an alarmingly high incidence of this complication during paediatric anaesthesia'.

The above opening paragraph to Chapter 30 of this book, and subsequent analysis of the factors upon which it is based, provide ample evidence that the safe management of anaesthesia in infants and children differs in certain important respects from standard practices in adults.

In this book, Dr. Smith has provided an outline of the specialized physiological and anatomical considerations upon which paediatric anaesthesia is based. From a vast practical experience he has succeeded most admirably in assembling more information concerning the basic response of children to anaesthesia than has been available previously in one book. A noteworthy feature is the provision of apposite information concerning pathology, surgery, and paediatric medicine, and the relationship which these subjects bear to anaesthesia in the total care of the patient.

This book is a most valuable addition to anaesthetic literature in providing such full information, both theoretical and practical, in a field where very few anaesthetists have the opportunity of obtaining extensive practical experience.

A.B.B.

ANIMAL EXPERIMENTAL RESEARCH

Experimental Surgery. Fourth edition. By J. Markowitz, *M.B.E.*, M.B. (Tor.), Ph.D., M.S. in Exp. Surg. (Minn.), J. Archibald, D.V.M., M.V.Sc., Dr. Med. Vet. (Giessen), M.R.C.V.S. and H.G. Downie, D.V.M., M.S. (Cornell), M.V.Sc. Pp. 800. 580 illustrations. 16 coloured plates. £5 0s. 0d. plus 2s. 9d. postage. London: Baillière, Tindall and Cox. Ltd. 1959.

This, the fourth edition of an original and valuable record of the field of experimental surgery, was the result of the happy collaboration of 3 scientists and veterinarians. The book's particular interest will be as a work of reference for those engaged in research, but every surgeon and student (graduate or not) will find it of great surgical and physiological interest.

The 35 chapters cover a great deal of work in a variety of fields, including tissue and organ transplantation and vascular surgery. Many surgical problems have been solved by means of experiments on dogs, but there is still a vast amount to be learnt. It is probable that life itself and adjustments to disease are very similar, if not identical, in all mammals, so experimental work of this nature will continue to be a source of information and instruction. On this, to a large extent, depends the advance of the science of medicine. The experimental work is discussed not only on the basis of the authors' own extensive experience, but with frequent reference to the work of other researchers in similar fields, whether positive results have been obtained or not.

Well-illustrated and documented with a comprehensive bibliography at the conclusion of each chapter, this is a most interesting book, well written and of considerable value either as a work of reference or as a textbook.

C.N.B.

SURVEY OF CONGENITAL HEART DISEASE

An Introduction to Congenital Heart Disease. By Leo Schamroth, M.B., B.Ch. (Rand), M.R.C.P.E., F.R.F.P.S. and Fay Segal, M.D. (Rand). Pp. ix + 116. 82 illustrations. 22s. 6d. net. Oxford: Blackwell Scientific Publications Ltd. 1960.

This book has been written as a guide to the medical student or practitioner who might be bewildered by the complexity of new knowledge in this important field. The basic principles and facts about the more important conditions are presented with clarity in summary form and there are numerous well-conceived diagrams and illustrations. The beginner is thus quickly orientated and offered a useful stepping-stone to deeper study of the subject. Details of investigative and surgical techniques are, with some justification, not given, but the serious student will be disappointed by the omission of a classification of congenital heart disease and many important references in the bibliography.

L.V.

BRIEWERUBRIEK : CORRESPONDENCE

THE CROSS-ROADS

To the Editor: Dr. Impey in his article 'The Cross-roads'[1] criticizes the medical profession generally and bases his arguments on an American lecture on medical ethics and a Leading Article in the *British Medical Journal*. America is a world of its own, their standards and conditions, not only in the practice of medicine but also in many other directions, differ from ours. What they do and how they do it cannot always be accepted by others. Standards and traditions differ in different countries. I find it difficult to accept that the fine traditions of medical practice in Britain have suddenly collapsed under the National Health Scheme.

Medicine is an honourable profession and it is no less so today than it was yesterday. We have always been subject to criticism. I do not think this is a bad thing. Unfortunately so much of it is ill-informed. I have been qualified for half a century and for over 40 years have practised as a consulting physician in South Africa. I have been associated with many medical men in my hospital as well as my private practice and have a high regard for their integrity and standards of professional behaviour. There has been the odd one who should never have been a doctor. Those are the ones that Dr. Impey and I, with others, had to deal with on the Medical Council. It is, however, unwise to generalize about the particular.

Many of the profession are in agreement with Dr. Impey that it is illogical for a man in full-time occupation to be allowed to engage in private practice. It could possibly be said that their skill was such that their work should not be confined to the poor and indigent. However in the Union and particularly in those towns where medical schools are situated we have a considerable number of men who are highly skilled, and it is doubtful, very doubtful, if they are not equally competent. I know of no other occupation in which a man who holds a full-time post is allowed a gainful side-line. The responsibility for permitting this lies on the shoulders of the universities. Members of university staffs are among the first to rise in their wrath if action by various authorities is in their opinion not moral, but in these cases they carefully avoid the problem on their own doorstep. If the universities, for one or other reason, are unable to increase the emoluments of the holders of the chairs, they should re-advertize the posts. With the wealth of highly skilled men that we have, there should be little difficulty in getting some who would be satisfied with the remuneration that is available. If this is not possible then, in spite of the modern trend for full-time posts, the universities will have to go back to part-time professors, for it is not possible to have champagne tastes on a beer income.

A. Bloom

268 Innes Road
Durban
14 February 1961

1. Impey, R. L. (1961): S. Afr. Med. J., **35**, 91.

THE CROSS-ROADS

To the Editor: In the issue of the *Journal* for 4 February 1961, my old friend Dr. Lance Impey[1] calls attention to the often heard suggestion that our profession is in danger of becoming commercialized. I think he and I agree with your previous Editorial article[2] quoted by him to the effect that the danger is not really imminent as far as our general body of practitioners is concerned. There are of course exceptions, but we should not allow our judgment to be affected by a few black sheep, and we must remember that black sheep are always more conspicuous than white ones. But times change and it is the future that we must take into consideration. It is more in the spirit of the Roman *caveant consules* that one should read and think over Dr. Impey's article.

Dr. Impey very logically links up his warning note with the question of the position of our clinical professors, with whom the whole question is connected in so many ways. It is largely these professors in whose hands the future of our profession lies. They are in a unique position to shape and mould the generation of practitioners that is coming on. They decide, very largely, the attitude, the outlook, the ethics, even the philosophy of the younger men and we must see to it that they can and will do this. It is unfortunate that somehow or other the word 'professor' in this connection has changed its meaning. 'Professor' is a title, indicating that the bearer devotes himself to teaching. Nothing more and nothing less. It is not a qualification in the medical sense, and it is certainly not a 'higher qualification'. Every clinical professor knows that among his fellow-specialists, and, may I say it, among his fellow general practitioners, there are many men and women who are just as competent and capable as he is to deal with any kind of ailment.

In simple words the problem is: is it a good thing for clinical professors to have private practices or not? I have already argued that what we may call the public, although they may look upon the professor as a sort of super-specialist, does not stand to lose anything if the professor would no longer be available to serve them.

There is such a lot to say for professors concentrating completely on teaching. Normally the professor will be attached to a big hospital and have a large number of serious cases under his care. There was a time when his work consisted of looking at tongues, feeling pulses, and a sort of all-round course of percussion, auscultation and palpation. It is very different now. Most of his patients have collected a large dossier of reports of various kinds, as we all know. If the professor does his duty well, his time will be fully occupied with studying all these reports and making them available in a digestible form for his students. Laboratories, X-rays and other aids have, in a way, facilitated his diagnostic task, but they have not made it less complicated. Then, there is the formidable selection of ways of treatment available nowadays, pharmaceutical, surgical, technical, psychological. Frequent interruptions will hamper his task, and isn't there a good chance that calls of private practice will, and in some cases must, receive preference? Or at least appear to receive preference? I shall not dwell on the justified irritation of students who see lectures cancelled because the professor is out on private practice. I think more of the example, of the impression given that a professor's chosen duty may be abandoned for the sake of financial gain or reputation. Do professors always fully realize how their juniors, if they are good juniors, try to mould themselves after the worthy example of the professor? His ethics become their ethics. A good professor cannot have time for private practice; if he tries to do it, his real job must suffer since time is a limited commodity. I have not yet really gone deeply enough into the amount of time and energy that must be spent in keeping abreast of the formidable amount of new work published in any speciality.

As Dr. Impey puts it so neatly, there is no obligation on any medical practitioner to take up teaching as his life's work. Teaching is a highly specialized job, calling for rather uncommon qualities, but to the right man it is also uncommonly rewarding. No man should become a teacher unless he has a desire to teach, and he should not take it up because of this curious and spurious imagined higher professional status. To the born teacher there is nothing more satisfying than the feeling that by example and work he has influenced a new generation to do its work well, without a first regard for fees; and in later life the lasting attachment of pupils to a good teacher is a constant influence for good on both sides.

I should like, without abandoning my subject, to say a few words about this vexed word 'research'. Somehow or other it has come to be expected from a professor that he should do 'research'. If he has the urge, let him do it; give him a chance, he will do it anyhow if he has that desire. A research worker researches because he cannot help himself. But to expect from a man whose heart is in teaching, that he shall be a research worker as well, is certainly absurd. It can, in such an enforced way, only lead to a multiplication of the somewhat sodden *réchauffés* already choking our journals. Let him rather spend his time reading and studying so that when one of his juniors reveals a capacity for research he can suitably guide and help him.

Summing up, by forbidding clinical professors private practice, nothing would be lost and enormous advantages would be gained. Of course, salaries should be adequate. It should not be impossible to find that extra couple of thousand rand to attract and satisfy the suitable man. But it should be given, not as one hears it said now, as compensation for what the man sacrifices in giving up private practice, but as a recognition of his intrinsic qualities as a teacher.

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11 February 1961

1. Impey, R. L. (1961): S. Afr. Med. J., **35**, 91.
2. Editorial (1960): *Ibid.*, **34**, 417.

THE SECRETARY'S COLUMN

To the Editor: I feel I cannot allow the statement[1] appearing in the *Journal* of 14 January in 'From the secretary's desk' to pass without comment, in view of the vote of censure passed on my Branch by Federal Council in October last year, for the manner in which the Border Branch criticized a statement by the Secretary in which he recommended to Council 'as a matter of extreme urgency that immediate recognition i.e. preferential tariff, be granted to all medical aid societies underwritten by S.A. Mutual, otherwise this society, which had always played the game in the past, would stop paying doctors fees direct and in full'.

In the item referred to above, the Secretary states that 'The main cause of the breakdown has been the persistence of the Society in sending to doctors cheques which were based on the tariff of fees for *approved* medical aid societies with the insistence that they be accepted "in full settlement"' and further 'and we would be failing in our duty to our members and colleagues, as well as to our patients, if we allowed ourselves to be dictated to by any big business concern in any way whatsoever'.

It would seem that, had the Chairman of Federal Council allowed discussion on the merits of the Border Branch's criticism — which discussion was denied — some justification for the criticism might have emanated.

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28 January 1961

[The quotation from the Secretary's letter, which was written on 1 July 1960, is not accurate — sentences which appeared in two separate paragraphs are joined together — Editor]

1. From the Secretary's Desk (1961): S. Afr. Med. J., **35**, 36.

MAXIMUM DOSE OF PROCAINE AND 'XYLOCAINE'

To the Editor: Drs. L. G. R. van Dongen and H. Glietenberg in their article[1] in the *Journal* of 28 January 1961, state that the generally accepted maximum dose of 'xylocaine' and procaine is 1 g., that is, 50 ml. of a 2% solution, 100 ml. of a 1% solution or 200 ml. of a 0·5% solution.

This dosage is incorrect. It is generally accepted that the

stronger the concentration the smaller the maximum dose. Thus the dosage is 200 ml. of a 0·5% solution, 75 ml. of a 1% solution and 25 ml. of a 2% solution.[2]

We use blocks for many operations in this hospital, with or without adrenaline, and have proved on a number of occasions that it is only when the maximum dosage for the required concentration is exceeded that convulsions occur.

In my opinion, even with adrenaline the maximum dosage, given above, should never be exceeded.

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Industria, Johannesburg
8 February 1961

1. van Dongen, L. G. R. and Glietenberg, H. (1961): S. Afr. Med. J., **35**, 75.
2. Morris, D. D. B. *In* Wylie, W. D. and Davidson, H. C. (1960): *A Practice of Anaesthesia*, pp. 813 and 815. London: Lloyd. Luke.

QUESTIONNAIRE ON NUTRITIONAL DISEASES

To the Editor: Last year the National Nutrition Research Institute addressed a questionnaire to each of approximately 6,000 practising doctors in the country. They were requested to list all cases of nutritional disease which they encountered in their practice during a period of 4 weeks in winter (May/June) and again in summer (November/December). The majority of medical men showed a lack of interest and a relatively poor response was received to the questionnaire. Fortunately some acceded to the request and some supplied more than the data requested. This was mainly the case where doctors practised at hospitals, clinics and mission stations.

At present completed questionnaires are still being received by the Institute and the statistical analysis of the figures has not yet commenced. It is, however, hoped that, in spite of the disappointing response by most of the doctors, valuable data will be obtained from those questionnaires which have been received. It is also expected to obtain interesting data in respect of the milk consumption by children of all races.

The NNRI wishes through the medium of your *Journal* to express its gratitude to all those who collaborated for their contributions. A final appeal is made to all those who have completed their questionnaires, but who have not returned them to the Institute, to do so without delay so that a critical study and interpretation of the data can be undertaken. It must again be stressed that where practitioners have not come across any cases of nutritional disease, this fact should also be brought to the notice of the NNRI.

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10 February 1961

URINARY CALCULI IN THE BANTU

To the Editor: The interesting paper by Dr. Wise and Professor Kark on 'Urinary calculi and serum-calcium levels in Africans, and Indians'[1] serves to confirm and underline the rarity of urinary calculi in the Bantu compared with its relative commonness in Indian and White populations. Its occurrence in Indians in India is patchy, but the cause of the condition and its irregular distribution is far from apparent. In New Delhi, which I recently visited, the condition of urinary calculi is common, especially in young people, yet every avenue of investigation so far has yielded negligible information of value. Investigations have included 'clinical appraisal with special reference to dietary and nutritional status, serum calcium, phosphorus, and alkaline phosphatase, citrate and mucoprotein levels of the blood, serum proteins, sodium and potassium, histological and histochemical study of the matrix of stones, and histological study of the wall of the bladder. A routine study of pH and bacteriology of urine was made'.[2] It is plausible, of course, to regard the occurrence of urinary calculi as one of the manifestations of chronic malnutrition, and many years ago McCarrison,[3] on the basis of his experimental studies, attached chief blame to a deficiency of vitamin A. Subsequent research, however, has provided no unequivocal evidence in support of this theory. The uncertainties in the aetiology of the condition place it in the same category as crypotogenic 'nutritional' heart disease, and possibly gynaecomastia as seen in Africans, both of which were once confidently attributed purely to faulty nutrition, whereas it has to be confessed that now we no longer have that assurance.[4] Our lack of knowledge presents a challenge, but one which is very difficult to meet; moreover, help is unlikely to be forthcoming from animal experimentation.

The suggestion that liver dysfunction, indirectly, may help to protect the Bantu from urinary calculi is interesting. Nevertheless, among many under-privileged populations elsewhere, which are characterized by equal or greater liver dysfunction, urinary calculi are common.

The suggestion that changes in drinking and other habits may have a bearing on the condition has been made in a recent epidemiological investigation of urolithiasis in Israel.[5] In 12 settlements with a total of 30,292 inhabitants there were 357 patients with urolithiasis (11·8 per 1,000), men being affected much more than women. There was a wide range of incidence in the settlements, from 1·6 to 34·1 per 1,000, according to age, country of origin and length of stay in the settlements. Among immigrants from the Middle East and North Africa there were 7·9 cases per 1,000, and in those from Europe, 27 per 1,000. A high incidence was associated with a low fluid intake, largely the result of transfer of drinking habits from European conditions, and low output of urine caused by large extrarenal fluid loss in conditions of heat and heavy manual labour.

I do not agree with the statement that 'the serum-calcium level is independent of the dietary intake' of the element. There is a good deal of evidence that populations habituated to a diet, *inter alia*, low in calcium, have relatively low serum-calcium concentrations, and *vice versa*.[6,7] To what extent these low levels are due to a low intake of the element or to other factors, is not known. As far as I am aware, low values have not been markedly elevated by ingesting calcium salts only. At this centre, however, we are seeking to throw some light on this aspect by determining serum-calcium levels in Bantu consuming similar diets but imbibing water of grossly different calcium concentration.

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Human Biochemistry Research Unit
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Johannesburg
14 February 1961

1. Wise, O. R. and Kark, A. E. (1961): S. Afr. Med. J., **35**, 47.
2. Ramalingaswami, V. and Aurora, A. L. (1960): *Fifth International Congress on Nutrition*, Panel 7, p. 1. Washington.
3. McCarrison, R. (1931): Brit. Med. J., **1**, 1009.
4. Walker, A. R. P.: Nutr. Rev. In press.
5. Frank, M., De Vries, A., Atsmon, A., Lazebnik, J. and Kochwa, S. (1959): J. Urol., **81**, 497.
6. Walker, A. R. P., Arvidsson, U. B. and Politzer, W. M. (1954): S. Afr. Med. J., **28**, 48.
7. Walker, A. R. P. (1958): Ann. N.Y. Acad. Sci., **69**, 989.

HEART DISEASE IN PREGNANCY

To the Editor: Mr. M. B. E. Sweet[1] has drawn attention to the dangers of sudden collapse and death of the cardiac patient in the third stage of labour and in the first 12 - 18 hours after delivery. Another dangerous period occurs a week after delivery, when the patient appears to be making good progress. Again, the chief cause of death is sudden heart failure; acute pulmonary oedema is not uncommon, and rarer causes are massive thrombosis of the deep pelvic veins, pulmonary embolism, and even mesenteric thrombosis. The last 3 conditions are nearly always associated with Caesarean section in Grade III or IV patients.

The prognosis of cardiac disease in pregnancy, particularly of mitral stenosis, may be very deceptive, since sudden death can take place at any stage of pregnancy. Many practitioners have experienced at least one or two such cases. A typical example is that of a young primigravida with a Grade I mitral stenosis. An experienced cardiologist had given very good prognosis when she was 2 months pregnant, yet 2 months later she died suddenly from acute pulmonary oedema.

Everyone agrees that cardiac patients should be under

constant medical supervision during pregnancy and that they should be admitted to hospital for complete rest before delivery — the time depending on the degree of compensation, namely, 2 - 4 weeks for a Grade I patient, 6 - 8 weeks for a Grade II patient and immediate admission for a Grade III or IV patient. In spite of all precautions there will still be some unexpected and unpredictable deaths in patients with well-compensated lesions, but that is a risk that has to be faced.

G. Maizels

203 Oasim
Port Elizabeth
14 February 1961

1. Sweet, M. B. E. (1961): S. Afr. Med. J., **35**, 87.

THE MEDICAL SERVICES PLAN

To the Editor: We are going to have, quite unneccessarily I am sure, an Extraordinary General Meeting of the Association, requisitioned 'with a view to implementing the Medical Services Plan in all the Provinces of South Africa'.

The trouble is, I fear, that most of the signatories of the requisition for the meeting are ignorant of the history and development of medical aid insurance generally and of the 'Plan' in particular.

It has been repeatedly stated and repeated again at the last Federal Council meeting at Vereeniging, when the Border Branch asked for an extension of the Plan to their Branch, that there was nothing to stop the directorate of the Plan from doing so. They only have to get a large number of doctors in an area sufficiently 'Plan minded' to become participating doctors at R20.00 a head, and a large number of the population to join as members. There are however snags in their way. They will not be able to keep their administrative costs proportionately as low as at present with largely voluntary aid. There is the ordinary administration to be done and then the constant monitoring of doctors' accounts, i.e. of both participating and non-participating doctors. This is going to cost time and trouble, and will voluteers always be forthcoming?

Then, the Plan has adopted an apparently easy way out of a possible financial loss through snowballing of claims, by doctors agreeing to reduce their accounts in the event of this happening. When the time comes for this to be done on a large scale, how long are the rank and file going to be satisfied? The difficulties for the Plan, if it is organized on a large scale, are only too obvious.

It is only by getting the Association as a whole to take over full responsibility for the Plan, running it in competition with our own long-encouraged and fostered medical aid societies and with the commercial insurers now coming into the field, that the Plan can be 'implemented in all the Provinces'.

Mr. Parvus has stated that the Directors of the Plan do not want to extend the 'present pilot area, until such time as the Association clarifies its policy with regard to commercial insurance companies . . . '. But they know the policy of the Association full well and we have been busy trying to come to an agreement *suitable to ourselves* with the companies for about a year now.

I submit that the directors are not satisfied with mere sponsorship and that they would be very pleased to hand the baby right over to the Association.

From the very beginning, when something like the Plan was first mooted, there was opposition to it because we already had encouraged a fairly large number of medical aid societies. Our policy was to foster their growth, because they were beneficial to medical practice and were a counter-blast to the formation of benefit societies or schemes, such as that at Iscor and Van Der Bylpark, which were very unpopular with us at the time. I think medical aid societies would have prevailed, if there had not always been the elements among ourselves and the public, who 'kill the goose that lays the golden eggs'.

In 1955 the Medical Association sent a man to Canada to study their 'Pre-payment' schemes. His trip and other services cost the Association over £2,000 and in the end there seems to have been nought for our comfort. In 1956 a Committee under the chairmanship of Dr. M. Shapiro was appointed and empowered to go ahead with trying to work out and establish an insurance scheme to be run as a non-profit company. Dr. Shapiro is recorded as having promised that it would cost the Association not a penny. In fact they would regard the close on £3,000 already spent, as a debt of honour, to be repaid when the Plan was on its legs.

It is unneccessary to go into the many debates on the matter since then, some of them very acrimonious. Suffice it to say that throughout, I believe, the majority of Federal Council was against risking the finances and the reputation of the Association in a scheme of running medical insurance ourselves. 'Sponsorship' was given, but it was made clear that the Association took no responsibility and would run no financial risk. To my mind we have already taken too much moral responsibility. For Heaven's sake let it end there.

It is not possible for us to 'implement' the Plan, unless we take it over. That, I am afraid, is the object of this General Meeting. Let us be wary.

No one should run away with the idea that Canada is a Utopia as far as medical insurance goes. In recent numbers of the *Canadian Medical Journal* articles were published by practising doctors, which do not by any means reflect complete satisfaction with medical insurance as it exists in Canada at the present time — Canada, which is held up to us as having such a wonderful Plan or Plans. Refer to the issues of their Journal for 15 and 22 October 1960, particularly the latter. It is stated, that their T.C.M. plans cover 4,000,000 people, while 4,000,000 are covered by commercial insurance and a further 750,000 by various local schemes sponsored by company or community organizations. It is amusing to read how the writer was more or less blackmailed to reduce his fees to an insured patient, only to find that he afterwards had to sign certificates to enable the patient to draw other insurances as well! The writer deplores the fact that there are these insurers outside the Medical Association, having *no contract* with it, and he shows how the doctor - patient relationship is often spoilt by this fact. That is true enough, but the point is that neither in Canada nor here is it possible to prevent outside insurers from entering the field. It is therefore better to try to reach an agreement with them, which is satisfactory to ourselves, than to try to compete with them.

Remember that other insurance companies besides The South African Mutual Medical Aid Society and SANSOM undertake medical insurance, but *not* on the *large scale*. Medical insurance is risky, and disputes with their clients do the companies more harm than good. Let the insurance companies fight it out with the public themselves, but let us stand firm and secure agreements with them, which protect ourselves as well as our patients. From both aspects the South African Mutual Medical Aid Society has fallen short of our requirements, so they are out of the running for our favours.

Let us continue fostering the medical aid societies and treat them sympathetically. From the days when Dr. Jack Gie, ably followed by Dr. Green, laid the foundation of our amicable relations with them, both sides have derived great benefits. If we take over the Plan, we are going to land ourselves in great difficulties. The disgruntled amongst the insured will assail us as an Association and a profession, while our own colleagues will have plenty of cause for complaint when they find that they are not getting the big fees so confidently held out to them by the originators of the Plan. Inter-doctor relationships in addition to doctor-patient relationship will go to Hades!

Much as I admire Dr. M. Shapiro's fertile brain and his energy, I am afraid his 'Plan' has come too late in the history of medical practice in South Africa, and it is much too risky and unnecessary for us to take it over now.

I invite all members, who agree with me, to send me their proxies for the Extraordinary General Meeting to be held in Bloemfontein on 8 March 1961.

Raymund Theron

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18 February 1961